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The Mathison Lectures.¹

LECTURE I.

SNAKE VENOMS AND ANTITOXIC IMMUNITY.

By C. H. KELLAWAY, M.D., M.S., F.R.C.P.,
Director of the Walter and Eliza Hall Institute
of Research.

MORE than fifteen years have elapsed since Gordon Clunes Mathison died of wounds shortly after being evacuated from Gallipoli—a tragic ending to a life of thirty-one years crammed with achievement and brilliant with promise. His was a personality of great vitality and of singular charm. He was deeply interested in people as well as in things. He had a

genius for friendship. His energy and enthusiasm were boundless and his interests manifold. He was a keen mountaineer, an enthusiastic fisherman and cricketer and a critical lover of art and music.

To physiology Mathison brought great gifts. His knowledge was wide and accurate, he had a clear insight into problems and an acutely critical judgement of the value of his own results and of those of other workers. His researches on the physiology of respiration in Professor Starling's laboratory at University College, London, had already marked him out as a man from whom great things were to be expected. Later he turned his attention to research in pathology and clinical medicine. He was intensely interested in the practical aspects of these subjects and regarded pathological and clinical data with the same critical insight which had marked his more academic researches. He was full of ideas and his enthusiasm was contagious. With

¹ Delivered in the Physiology Lecture Theatre of the University of Melbourne, November 11 and 18, 1930.

his gift of lucid exposition and the capacity to attract others to research and find problems for them, he could not have failed to do great things for medical research in Australia.

I am greatly privileged to deliver the second of the lectures founded at this university in his honour by a number of Mathison's friends. In the first of these Professor W. A. Osborne discussed some new aspects of the physiology of respiration. For the present lectures I was not able to select a subject in such direct continuity with Mathison's own work. There is, however, continuity in another sense, for Mathison was the first director of the research laboratories at the Melbourne Hospital and had he survived the war would have been the director of the Hall Research Institute.

I propose in this and the following lecture to present the results of some investigations on immunity to snake venoms which have been carried out at the Institute during the last two years.

The first lecture will be concerned with the specificity of active immunity to snake venoms and the evidence for cellular immunity, and the second with the nature of the immunity of venomous snakes to their own venoms. In most of this work I have had the able assistance of Miss F. E. Williams. Miss Freeman has carried out all weighing of venoms, much of it on the micro-balance, and Mr. Tom Eades and Mr. Donald Thomson have helped me in handling reptiles.

The dissections of the vascular system of the death adder which are used in illustrating some points in the second lecture are the work of Mr. Donald Thomson and the excellent drawings of these were done for me by Mrs. Thomson.

I am greatly indebted to Dr. F. M. Burnet for permission to use protocols from some of his unpublished experiments.

The expenses of these researches have been defrayed by a grant from the Commonwealth Department of Health.

THE SPECIFICITY OF ACTIVE IMMUNITY TO SNAKE VENOM AND THE EVIDENCE FOR CELLULAR IMMUNITY.

The extensive literature on passive immunity against snake venoms has been admirably surveyed by Noguchi⁽¹⁾ and by Marie Phisalix⁽²⁾ and I do not propose to review it again here. It is sufficient to remark that the experiments of Martin,⁽³⁾ Lamb,⁽⁴⁾ Flexner and Noguchi^{(5) (6)} and Tidswell⁽⁷⁾ have demonstrated the high degree of specificity exhibited by monovalent antivenines in the neutralization of snake venoms and that the more recent work of Arthus,⁽⁸⁾ of Houssay and his colleagues,⁽⁹⁾ of Brazil⁽¹⁰⁾ and of Vellard⁽¹¹⁾ has modified conclusions based on this earlier work to the extent of admitting that venoms of closely related species may exhibit non-specific reactions.

The specificity of active immunity has, however, been little studied. Phisalix⁽¹²⁾ found that guinea-pigs immunized against viper venom were not protected against the venom of the cobra and, conversely, that animals immunized against cobra

venom were not protected against viper venom. These two snakes are, however, widely separated zoologically and their venoms are totally different in the toxic effects which they produce.

In another place⁽¹³⁾ I have detailed the results of an investigation of the specificity of active immunity in guinea-pigs and I propose here to utilize some of these results and compare them with later studies by Miss Williams and myself on the specificity of active immunity in rabbits in which the degree of humoral immunity was controlled by protection experiments in guinea-pigs.

A necessary preliminary to the appreciation of any investigation involving the use of the venoms of closely allied snakes is a knowledge of the "toxic constitution" of the venoms and of the serological relationships of the reptiles yielding them.

The venoms utilized in both sets of experiments were those of the death adder (*Acanthophis antarcticus*), tiger snake (*Notechis scutatus*) and copperhead (*Denisonia superba*) and in the experiments on guinea-pigs the venoms of the black snake (*Pseudechis porphyriacus*), the cobra (*Naia naia*) and the daboia (*Vipera russelli*) were used in addition. The venoms of two other snakes representing other genera of Australian elapines were also used for testing—those of the giant brown snake, *Oxyuranus macleynani*, and the common brown snake, *Demansia textilis*.

The venoms of these snakes are highly complex in their toxic reactions. That of *Demansia textilis* is powerfully neurotoxic, has a tendency to produce hæmorrhages and causes hæmolysis. If injected intravenously, it causes coagulation of the blood. The venom of *Pseudechis porphyriacus* is also neurotoxic, but is powerfully hæmolytic, causes striking hæmorrhagic effects and has a powerful coagulant action. The venom of *Denisonia superba* is almost purely neurotoxic in action, it has a feeble coagulant effect in very large doses *in vitro*; no coagulant action can be demonstrated *in vivo*, indeed it has some anticoagulant action. It is hæmolytic in most species of experimental animals and it has also some hæmorrhagic action, though this is relatively slight. The venom of *Notechis scutatus* is powerfully neurotoxic, coagulant *in vivo* and *in vitro* and has less strong hæmorrhagic and hæmolytic tendencies than are observed with the venom of *Pseudechis*. The venom of the death adder is powerfully neurotoxic and in some species, particularly the guinea-pig, exhibits striking hæmorrhagic effects. Like that of *Denisonia superba*, it has only a very feeble coagulant action *in vitro* and none *in vivo*, where it has a slight anticoagulant activity. Its hæmolytic activity is feeble. The venom of *Oxyuranus macleynani* is powerfully neurotoxic, coagulant *in vivo* and *in vitro*, but only feebly hæmolytic and hæmorrhagic in tendency. The venom of the cobra is neurotoxic with a powerful peripheral action. It is also more strongly hæmolytic than any of the Australian elapine venoms and exhibits a striking hæmorrhagic tendency, particularly in the guinea-pig. It has no coagulant action, but

on the contrary is strongly anticoagulant. The venom of the daboia is a typical viperine venom, powerfully coagulant, with very little neurotoxic activity, but capable of producing marked local effects at the site of injection.

The very close serological relationship of some of these species has been shown by Miss Williams and myself⁽¹⁴⁾ by the use of the precipitin and complement fixation reactions of antisera obtained from rabbits. The classical researches of Nuttall⁽¹⁵⁾ showed that it is possible to differentiate members of the classes, orders and even families of the animal kingdom by the precipitin reactions of their sera. Strong antisera gave crossed reactions with heterologous sera and these non-specific reactions were stronger with species which were closely related zoologically than with more distantly related species. Graham Smith extended Nuttall's work, which was mainly concerned with mammalian and avian sera, enlarging his results by observations on the sera of reptiles, amphibians, fish and crustaceans.

Our studies showed that while the strongest precipitin reactions were obtained with the sera of the reptiles used to produce antisera, crossed reactions occurred with all the other sera from Australian proteroglyphous elapine snakes which were tested and, in the case of strong antisera, with those of *Dendrophis punctulatus* (aglyphous) and of *Python spilotes* (family Boidæ) also. The degree of reaction afforded no indication of the zoological order of the proteroglyphous elapines which, according to Boulenger,⁽¹⁶⁾ is: *Demansia*, *Pseudechis*, *Denisonia*, *Notechis*, *Acanthophis*, an order which was confirmed by Hamilton Fairley's studies⁽¹⁷⁾ of the osteological characters and biting mechanism of snakes in these genera. The results of complement fixation were only slightly more specific than those of the precipitin test and absorption experiments enabled us to obtain antisera with strict species specificity in only a few instances. Further studies on agglutination of the red blood cells in these species demonstrated their extremely close blood relationship.

Results of Experiments on Guinea-Pigs Actively Immunized against Tiger Snake Venom.

Forty guinea-pigs were immunized by subcutaneous injections of increasing amounts of tiger snake venom at weekly intervals during seven months, at the end of which time they were immune to at least fourteen certainly lethal doses of this venom. During the testing of these guinea-pigs with other venoms, which occupied about a month, injections of 0.15 milligramme of tiger snake venom were administered at weekly intervals. The guinea-pigs were tested seven to ten days after these injections. By using a number of guinea-pigs to test each dose it was hoped to eliminate individual variations in immunity. In Table I there are set out the results of the investigation of this series of animals by the subcutaneous injection of various venoms.

TABLE I.
Non-Specific Immunity of Guinea-pigs Sensitized to at least Fourteen Certainly Lethal Doses of Tiger Snake Venom.

Type of Immunity.	Dosage.
Immunity to copper-head venom ..	12 certainly lethal doses
Immunity to death adder venom ..	Less than 1.5 certainly lethal doses
Immunity to brown snake venom ..	Less than 2.5 certainly lethal doses
Immunity to black snake venom ..	Nil
Immunity to giant brown snake venom ..	At least 8 certainly lethal doses
Immunity to cobra venom ..	Nil

Striking non-specific protection was exhibited against two powerfully neurotoxic venoms, those of *Denisonia superba* and *Oxyuranus macleodensis*. Against the venoms of the brown snake and death adder whose lethal action, when injected subcutaneously, depends also upon neurotoxin, there was some slight degree of protection, but to the venom of the black snake, in which the killing effect is largely hæmotoxic, no protection was observed. Cobra venom is also powerfully hæmotoxic in the guinea-pig and it is uncertain whether the complete failure of protection against this venom is due to this fact or depends on the more distant zoological relationship of this reptile to the tiger snake.

The non-specific passive protection afforded by a univalent tiger snake antivenine, which was given me by Dr. F. G. Morgan, Director of the Laboratories Division of the Commonwealth Health Department, yields an interesting comparison. One cubic centimetre of this serum protected against forty-eight certainly lethal guinea-pig doses of tiger snake venom, against more than six certainly lethal doses of *Oxyuranus*, against about three certainly lethal doses of copper-head, against less than one certainly lethal dose of *Pseudechis australis*, and showed no significant protection against the venoms of the brown snake, black snake and death adder.

Now while we may not assume that the antibody response is quantitatively similar in the horse and guinea-pig, this comparison suggests that active immunity is somewhat less specific than passive immunity.

Results in Guinea-Pigs Actively Immunized against Copper-Head Venom.

A series of thirty-seven survivors of the immunization to this venom were used to test non-specific immunity by subcutaneous injection, with the results set out in Table II.

TABLE II.
Non-Specific Immunity in Guinea-pigs Immunized to at least Nine Certainly Lethal Doses of Copper-head Venom.

Type of Immunity.	Dosage.
Immunity to tiger snake venom ..	4 to 6 certainly lethal doses
Immunity to giant brown snake venom ..	Less than 2 certainly lethal doses
Immunity to black snake venom ..	Nil
Immunity to death adder venom ..	Less than 1.5 certainly lethal doses
Immunity to brown snake venom ..	About 1.5 certainly lethal doses
Immunity to cobra venom ..	Nil

Immunization with a venom which is almost wholly neurotoxic in action, afforded striking non-

specific protection only to the venom of the tiger snake. This result must be held to indicate the close zoological relationship of the tiger snake and copper-head, as well as the similarity of their venoms in predominant neurotoxic action when injected subcutaneously.

Results in Guinea-Pigs Immunized with the Venoms of the Death Adder, Cobra and Daboia.

There were eight survivors of the immunization to death adder venom and though the largest dose they had received was equivalent only to two certainly lethal doses of venom, it is possible that they were capable of withstanding much larger doses. These animals were found to be immune to twelve certainly lethal doses of tiger snake venom and to more than eight certainly lethal doses of copper-head venom. In contrast to these results were those in guinea-pigs immunized to the venom of the cobra to a similar level to that obtaining in the death adder series and in which there were only fifteen survivors. These showed no appreciable protection to the venoms of the copper-head or death adder and only a trivial degree of protection against tiger snake venom. The results in these two series demonstrate the importance of close zoological relationship in determining non-specificity.

Forty-guinea-pigs immunized to large doses of daboia venom also developed no appreciable protection to the venoms of the copper-head, tiger snake, death adder and cobra. In this immunization to a viperine venom, the absence of any non-specific protective effect against colubrine venoms was to be expected.

Results of the Intravenous Injection of Tiger Snake Venom in Guinea-Pigs Immunized with Tiger Snake, Copper-Head, Black Snake and Daboia Venoms.

The testing venom contains a powerful thrombin and of the venoms used for immunization those of the black snake and daboia also contain a thrombin. Copper-head venom, on the contrary, has an anticoagulant action. The tiger snake immune animals were found to resist about twelve intravenous certainly lethal doses of tiger snake venom; the copper-head immune animals withstood four intravenous certainly lethal doses. The black snake immune guinea-pigs were protected only to a very slight extent and the daboia immune guinea-pigs not at all.

Protection against the thrombin of tiger snake venom depends therefore on the specific nature of the venom used for immunization rather than on its content of thrombin. Protection was obtained by immunization with a closely allied venom which contains no thrombin, but not with a viperine venom, despite its powerful coagulant action.

In both active and passive immunity two factors appear to determine the degree of specificity: (i) The zoological relationship of the reptiles and (ii) the nature of the venom—its toxic constitution, and particularly that toxic action which is dominant in causing a fatal result.

The results of the experiments just outlined suggest that active immunity exhibits a less strict specificity than passive. Close zoological relationship between the reptiles yielding the venoms, particularly if these are of similar "toxic constitution," may determine a by no means inconsiderable degree of non-specificity in active immunity. In the experiments made by subcutaneous injection the presence or absence of thrombin did not influence the result except in so far as it operated in delaying the absorption of coagulant venoms.

The most striking feature in these experiments was the protection afforded against the venoms of the tiger snake and copper-head by immunization with death adder venom, though immunization with tiger snake and copper-head venoms yielded no appreciable protection against the venom of the death adder. This is probably to be explained by the presence of two antigenic substances in death adder venom, one associated with a "hæmorrhagin" and the other with a neurotoxin closely related to the neurotoxins of tiger snake and copper-head venoms.

The recent experiments of Vellard⁽¹¹⁾ on passive immunity to viperine venoms are of interest in this connexion. He used a monovalent serum prepared against the venom of *Crotalus terrificus*, a polyvalent "anti-bothropique" serum prepared against various species of *Lachesis*, a monovalent serum prepared against *Lachesis jararaca*, a polyvalent serum prepared against European viperine venoms and a serum prepared at Tokyo (probably anti-lachesis and anti-anastradus). These were tested against the venoms of *Crotalus terrificus*, *Lachesis jararaca*, *Lachesis lanceolata*, *Lachesis flavoviridis* and *Vipera aspis*, using intravenous injection in the pigeon and *in vitro* tests of coagulant, anticoagulant and proteolytic power. Vellard concluded that the sera were highly specific against the venom or venoms used in their preparation, but observed that the "anti-bothropique" and the anti-jararaca sera gave higher protection in lethal doses, though not in milligrammes of venom against the venom of *Crotalus* than against the *Lachesis* venoms used in their preparation. This was due to the great activity of the venom of *Crotalus terrificus*, the lethal dose in the pigeon being 0.001 milligramme, while those of *Lachesis flavoviridis* and of *Lachesis lanceolatus* were 0.3 and 1.5 milligrammes. Here is a phenomenon similar to that observed in the case of guinea-pigs actively immunized with death adder venom and exhibiting a higher immunity, as judged by numbers of lethal doses, against the venoms of the tiger snake and copper-head. This curious result may therefore be partially explained by the higher activity of the venoms of the tiger snake and copper-head in the guinea-pig, their lethal doses per 100 grammes being 0.002 and 0.007 milligramme, compared with 0.015 milligramme for the venom of the death adder.

At this stage it is necessary to recall the fundamental work of Glenny⁽¹⁸⁾ and his colleagues on the difference between the antibody response to an

initial injection of antigen and that which follows a dose of specific antigen in an animal already immunized. Glenny and Südmerson⁽¹⁹⁾ gave a horse two doses of diphtheria toxin-antitoxin mixture at an interval of fourteen and a half weeks. The response to the primary stimulus was slight and gradual and that to the secondary stimulus was striking and relatively rapid.

Dr. Burnet has recently carried out a very instructive experiment which shows how the extent of the secondary response depends upon the intensity of the initial stimulus. The figures in Table III show clearly the difference in response to the same secondary stimulus following primary stimuli of graded size.

On the same day varying doses of staphylococcus toxin were injected into five rabbits and eighteen days later a dose of a toxoid prepared from the same toxin caused changes in the hæmolytic titre of the sera of the animals which were related in extent to the size of the original dose of toxin.

Two factors may conceivably operate in causing the non-specificity observed in guinea-pigs actively immunized with snake venoms to be more striking than that observed in passive protection experiments with immune sera. The closely allied venoms of similar toxic constitution may be able to function as specific secondary stimuli or there may be an acquired general tissue resistance (cellular immunity). It is unlikely that any truly non-specific secondary stimulation could result in sufficient antibody response to account for the observed protection to closely allied venoms, but if the venoms are nearly enough allied to act as specific secondary stimuli, time relationships must to a large extent determine the protective value of this reaction in the actively immune animal. Can antibody be produced by a secondary stimulus sufficiently rapidly to account for any significant additional protection? Dean and Webb⁽²⁰⁾ have shown that the secondary precipitin response to horse serum in an immune rabbit takes seven days to become maximal and during the first two days no appreciable response is to be observed. It must, however, be borne in mind that the antigen introduced as a secondary stimulus must be neutralized during this early period before any rise can be detected, and that if antibody production be insufficient to effect this neutralization, a fall in titre (in an animal in which some free antibody was already present at the time of the injection) should be observed. It

will later be shown that such a fall of titre in immune animals actually does occur.

Now the death times following the injection of snake venom depend on the size of the dose (Acton and Knowles,⁽²¹⁾ Glenny⁽²²⁾) and also upon the nature of the venom. In the case of the predominantly neurotoxic venoms, tiger snake, copper-head, brown snake and giant brown snake, the death times are on the average prolonged, both in rabbits and guinea-pigs, when only a single certainly lethal dose is given subcutaneously, some animals dying on the second, third or fourth days. With black snake venom in both the rabbit and guinea-pig, though occasionally death is delayed, for the most part it takes place in less than fifteen to eighteen hours. In the guinea-pig and rabbit with death adder venom, death is rapid, nearly all the animals dying on the first day. It is evident then that the action of a closely allied venom as a secondary specific stimulus is more likely to be efficient as a protective mechanism following injection of the venoms of the tiger snake, copper-head, brown snake and giant brown snake than those of the black snake and death adder, and it is possible that the higher non-specific protection observed against the former group of venoms may in part be dependent upon this consideration.

A study of the active immunity to these venoms in another species of experimental animal would, it was thought, bring out fresh differences. In the rabbit, death adder venom has not nearly so striking a hæmorrhagic effect as in the guinea-pig, and it is possible to titrate the humoral immunity of these larger animals at the time of testing with other venoms, and to ascertain whether closely allied venoms can act as secondary stimuli. Further, it was hoped that by blocking the "reticulo-endothelial" system (by removal of the spleen and injection of trypan blue or india ink) indirect evidence might be obtained in regard to the part played by cellular immunity in determining the degree of resistance observed to the injection of closely allied venoms. We therefore immunized three groups, each of about a dozen young healthy domestic rabbits, with the venoms of the tiger snake, copper-head and death adder.

Rabbits Immunized with Tiger Snake Venom and Tested with the Venoms of the Copper-Head and Death Adder.

In the rabbits immunized with tiger snake venom immunization was commenced in August, 1929, and the highest subcutaneous dose, 2.6 milligrammes,

TABLE III.
To show how the Size of the Primary Stimulus affects the Secondary Response.

Rabbit.	Toxin, October 3, 1930.	Titre, October 13, 1930.	Toxoid, October 21, 1930.	Titre.	
				October 27, 1930.	October 29, 1930.
4	0.1 c.cm. subcutaneous	<4	0.2 c.cm. subcutaneous	120	400
2	0.01 c.cm. subcutaneous	<4		48	120
1	0.001 c.cm. subcutaneous	<4		24	80
6	0.0001 c.cm. subcutaneous	<4		0	0
11	0.0001 c.cm. intravenous	<4		0	0

was reached after eight and a half months. The animals were then immune to at least twenty certainly lethal doses of this venom. After this time, since some of them had local subcutaneous abscesses, a course of intravenous injections was given. They received their last intravenous dose, 0.11 milligramme, on June 23, 1930, but were not used for the observations recorded here till August and September, 1930. The animals were thus allowed to lose much of their "humoral" protection before being studied. The degree of humoral protection at the time of testing was observed by titration of the serum, mixtures with venom being used which were allowed to stand for one hour before subcutaneous injection into guinea-pigs of about 250 grammes weight. The rabbits gained weight regularly during immunization and had no symptoms following the injections. There were no deaths. Though they all received the same treatment, there was some variation in the degree of humoral immunity reached.

A week after the last injection the titres of the sera of the first three rabbits were 0.15, 0.09 and 0.15 milligramme per cubic centimetre. A week later the titres of the sera of rabbits numbers 2, 3 and 4 were 0.056, 0.11 and 0.07 expressed as milligrammes of tiger snake venom neutralized by one cubic centimetre of sera. A number of observations at different times on the sera of the remaining rabbits showed variations of a similar order.

Even when the titre of the sera to tiger snake venom had fallen to 0.02 milligramme per cubic centimetre, the rabbits were immune to fifteen certainly lethal doses of tiger snake venom and to three certainly lethal doses of the venom of the copper-head. Rabbits of higher titre (0.15 milligramme of tiger snake venom per cubic centimetre) were immune to three certainly lethal doses of death adder venom.

Passive protection by the sera to tiger snake and copper-head venoms was in the proportion of about 2:1, so that, having regard to the certainly lethal doses of the venom in the rabbit (0.045 and 0.5 milligramme per kilogram respectively), the proportional protection in the actively immune animal, if this depended entirely on the antibody already present in the plasma, should have been about 22:1. A much higher protection was observed in the whole animal to copper-head, the proportion being 5:1.

The proportional protection of the sera to tiger snake and death adder venoms was about 15:4 or, taking account of the lethal dosage of these venoms in the rabbit (0.045 milligramme and 0.15 milligramme per kilogram), 25:1. It is not, however, fair to compare the protection of these rabbits to tiger snake and death adder venom, since by chance only high titre rabbits were used in testing immunity against this latter venom. These, however, were probably not immune to much more than twenty certainly lethal doses of tiger snake venom and therefore the protection against three certainly lethal doses of death adder venom is probably much too high to be due to antibody already present in

the plasma and is probably accounted for either by the capacity of death adder venom to act as a specific secondary stimulus or by cellular immunity. There are two possible fallacies which should be kept in mind in applying this method of comparison: (i) The number of animals used for testing may, in view of the variation in their humoral immunity, be insufficient to provide an accurate estimate of the immunity of a group of animals to two or three venoms and (ii) in the special case of animals immunized against tiger snake venom the proportional protection to the venoms of the copper-head and death adder may appear to be too high, because intravascular thrombosis may occur following the subcutaneous injection of large doses of tiger snake venom, and our estimate of immunity to this venom may be too low.

Rabbits Immunized with Copper-Head Venom and Tested with Tiger Snake and Death Adder Venoms.

The rabbits immunized with copper-head venom received a course of injections twice weekly, starting in August, 1929, and on May 1, 1930, reached a maximal subcutaneous dose of 7.6 milligrammes—equivalent to about five certainly lethal doses of this venom. Two died during the course of the immunization and three were used for other purposes. Only seven were available for the final tests. The immunizing injections were carried on till August 4, 1930, but during the last two months weekly intravenous injections of 1.5 milligrammes of copper-head venom were given to allow time for the treatment and sound healing of abscesses caused by earlier subcutaneous injections.

An interesting, though unintentional, experiment was made with these animals on March 3, 1930, when their average weight was 2.66 kilograms. They were then given 2.0 milligrammes or about five certainly lethal doses of death adder venom subcutaneously. One or two animals appeared a little upset by the injection for an hour or two, but none of them had any serious symptoms. At this stage they were presumably immune to about two certainly lethal doses of copper-head venom subcutaneously, since their last dose had been 2.4 milligrammes.

On August 11, 1930, the titre of the sera of the rabbits to copper-head venom ranged between 0.07 and 0.22 milligramme per cubic centimetre. The variation in immunological response to the same treatment is therefore considerable.

It is of interest to compare the protective power of the sera of these rabbits against the three venoms. Though this could be done only with a first approximation to accuracy, the proportional protection by the sera from different rabbits to any pair of venoms was in fair accord and the proportional protective power calculated from the titres in milligrammes per cubic centimetre was:

Copper-head.	Death adder.	Tiger snake.
11	1	3

Having regard to the certainly lethal dosages in milligrammes per kilogram (for the rabbit 0.5, 0.15

and 0.045 milligramme respectively) the proportional protective power was:

Copper-head.	Death adder.	Tiger snake.
1	0.3	3

Now the actual protection observed in the experimental testing was to five certainly lethal doses of copper-head, eight of tiger snake and four and a half to six of death adder venom, a proportion of:

Copper-head.	Death adder.	Tiger snake.
5	5	8

The active immunity to death adder venom is therefore greater than would be expected from the titre of the serum to this venom. It is clear then that either the death adder venom must act as a specific secondary stimulus or there must be some degree of acquired tissue immunity to this venom. Usually, if the dose of death adder venom is much above the certainly lethal dose, death takes place rapidly in four or five hours or less, and it is questionable whether the response to a secondary stimulus could occur sufficiently rapidly to explain the observed protection.

This high immunity to death adder venom does not depend on the few final observations, for all these rabbits were the victims of the unintentional experiment already referred to, and survived the injection of five certainly lethal doses of death adder venom at a time when they were probably only immune to somewhat more than two certainly lethal doses of copper-head venom. On account of this mistake these animals were not available for testing the action of death adder venom as a specific secondary stimulus.

The protection to tiger snake venom was, however, not so great as appeared to be indicated by the protective titre of the serum. This probably depended on the absence of protection to the thrombin of tiger snake venom, since deaths from intracardiac thrombosis tended to follow the subcutaneous injection of multiple lethal doses of this venom.

Rabbits Immunized against Death Adder Venom and Tested with Copper-Head and Tiger Snake Venoms.

The rabbits immunized to death adder venom were thirteen in number, and since immunization in the guinea-pig had been attended by numerous fatalities, the process was carried on very carefully. Immunization was commenced in August, 1929. On May 1, 1930, they received a dose of 3.8 milligrammes (about eight certainly lethal doses) of venom subcutaneously. After this a course of intravenous injections was given, reaching 2.7 milligrammes on August 11, 1930, when the injections were discontinued. Two animals died immediately after being injected on May 15, 1930, possibly because the injection rate was somewhat too rapid. Otherwise there were no fatalities during this immunization. The animals put on weight regularly and were in excellent condition throughout. A week after the last injection the titre of the sera of these rabbits estimated by protection experiments in guinea-pigs varied from 0.033 to 0.1 milligramme of

death adder venom per cubic centimetre. Some of these animals were doubtless immune to more than eight certainly lethal doses of death adder venom at this stage, but by the end of the period of testing, which commenced about a month later and lasted three weeks, none of the survivors withstood more than eight certainly lethal doses of this venom. By this time the titre of the serum to death adder venom had fallen to less than 0.015 milligramme per cubic centimetre.

Comparison of the protective power of the sera of these rabbits to different venoms gave a proportion based on the neutralizing power of the sera in milligrammes per cubic centimetre of:

Death adder.	Copper-head.	Tiger snake.
2	1	1

Expressing this result in terms of the lethal doses of the venoms for the rabbit we have the proportion:

Death adder.	Copper-head.	Tiger snake.
9	2	22

as the measure of the immunity of the animals if it were wholly dependent on antibody already present in the plasma.

Now the direct determination of the protection of the whole animal, based, it is true, on only a small number of animals, showed the actual protection by active immunity to be in the proportion:

Death adder.	Copper-head.	Tiger snake.
8	3.5	8

It is evident that the protection of the whole animal to copper-head venom is greater and that to tiger snake venom less than would be expected if immunity depended only on antibodies already present in the plasma.

The result in the case of tiger snake venom is almost certainly dependent upon the presence of thrombin in this venom (to which death adder immunization confers no immunity) and upon the rapidity with which venom in amount sufficient to cause intravascular thrombosis is absorbed into the blood stream when large doses are employed. Two animals which received ten certainly lethal doses of tiger snake venom died in ten minutes and in an hour with intracardiac thrombosis.

The protection to copper-head venom may be due to one of two causes, either to an acquired immunity of the tissues (cellular immunity) or to the production of fresh antibody, the copper-head venom being sufficiently nearly allied to death adder venom to act as a specific secondary stimulus. It is doubtful, however, in this case whether sufficient time would be afforded for a secondary rise in the titre of antibody to be effective unless there were also present some acquired immunity of the tissues.

The Response of Immune Animals to Secondary Stimuli.

The fall of titre following the cessation of injections of tiger snake venom and its rise following an injection of the same venom are well illustrated by the results in tiger snake immune rabbit number 4. By July 7, 1930, when the first test was made, the titre had already fallen probably by about half

to 0.07 milligramme per cubic centimetre. A month later it was 0.026 milligramme per cubic centimetre, and on September 18, 1930, it was between 0.012 and 0.017 milligramme per cubic centimetre. The injection of fifteen certainly lethal doses of tiger snake venom caused a further fall within two days to less than 0.008, but five days after the injection the titre had risen to 0.22 milligramme and eight days after the injection it had risen higher still.

Now though, as is well recognized, an unrelated antigenic substance cannot act as a secondary stimulus except possibly to a trivial extent, and causes only rise of its own antibody in the serum, owing to the very close relationship of these venoms it was to be expected that they might react as secondary stimuli in rabbits immunized with other venoms. In the tiger snake immune rabbits this was clearly the case following the injection of both death adder and copper-head venom.

In tiger snake immune rabbit number 11, on September 2, 1930, the titre to death adder venom was 0.017-0.02 milligramme per cubic centimetre and that to tiger snake venom 0.15 milligramme per cubic centimetre. Six days after the injection of three certainly lethal doses of death adder venom the titre to death adder venom was 0.05 milligramme and to tiger snake venom 0.22 milligramme per cubic centimetre. A similar phenomenon is seen in tiger snake immune rabbit number 10, injected with two and three-quarter certainly lethal doses of copper-head venom. Three weeks before the injection the titre of the serum was 0.016-0.022 milligramme of tiger snake venom and less than 0.015 milligramme of copper-head, and on the day of the injection the titre must have been considerably lower. Five days after the injection the titre was 0.45 milligramme of tiger snake venom and more, probably much more, than 0.032 milligramme of copper-head venom per cubic centimetre. In tiger snake immune rabbit number 3, on August 28, 1930, the titre of the serum was 0.018-0.022 milligramme of tiger snake venom and less than 0.011 milligramme of copper-head. Following the injection of one and three-quarter certainly lethal doses of copper-head venom the titre rose on the fourth day to 0.11 milligramme of tiger snake and to more than 0.06 milligramme of copper-head, and on the eighth day to more than 0.5 milligramme of tiger snake venom and more than 0.45 milligramme of copper-head.

These results make it certain that the closely allied venoms not only act as specific secondary stimuli, causing a rise in the titre to tiger snake venom, but also lead to a greater proportional rise in the titre to the venom used for this injection. They would serve to explain the relatively high protection observed in these actively immunized animals to venoms other than that used for immunization, if it could be shown that sufficient time were afforded for the production of antibody.

In the examples quoted above it was not possible to obtain closely spaced observations, because for satisfactory titration large quantities of sera were

needed at each bleeding (about twenty cubic centimetres of blood). Such large bleedings repeated at frequent intervals themselves tend to increase the titre of antibody. These and other observations show that an initial fall of circulating antibody follows the subcutaneous injection of venom and that the time relations are much the same as in Dean's experiments with horse serum. Dr. Burnet has carried out an accurate time curve for the specific secondary response to staphylococcal toxin, which shows general agreement with the other data already quoted.

Though the response to a secondary closely related stimulus in these animals immunized with various venoms undoubtedly plays some part in determining their resistance to the subcutaneous injection of other closely related venoms, it is doubtful whether the time necessary is sufficient to account for the phenomena observed unless there is also some acquired immunity of the tissues.

Cellular Immunity.

Rabbits immunized against the venoms of the tiger snake, copper-head and death adder are not suitable for use for the direct investigation of cellular immunity by the methods utilized by Gunn and Heathcote,⁽²³⁾ namely, by the study of the reaction of perfused organs to the venoms. None of these venoms has any sufficiently striking action on the isolated perfused heart or upon the isolated auricle preparation.

We therefore made no attempt to investigate the presence of cellular immunity directly by the use of these immune animals, but instead sought to obtain indirect evidence by excising the spleen and blocking the reticulo-endothelial system in some of them before injecting a sublethal dose of one or other of these venoms.

Our results which will be recorded in detail elsewhere, were not decisive in experiments upon rabbits immunized with tiger snake and copper-head venoms, but in the experiments upon rabbits actively immunized with death adder venom the exclusion or delay of a secondary stimulus by these procedures appeared to make no difference to the active immunity of the animals to other venoms. In these animals at least it appeared probable that some acquired cellular immunity must be postulated.

Other evidence is available which points in the same direction. The rabbits immunized with death adder venom were immune to eight certainly lethal doses of this venom, to about three certainly lethal doses of copper-head and eight certainly lethal doses of tiger snake venom, yet the titre of their serum at the end of immunization appeared to be too low wholly to account for their immunity. Furthermore, Dr. F. G. Morgan's experimental immunization of horses has shown that while a very substantial active immunity may be built up to death adder venom in this animal, the serum titrated by protection experiments in guinea-pigs has only a trivial protective power (one cubic centimetre at no time neutralizing more than four certainly lethal guinea-

pig doses). These low titres are not to be explained by a humoral response to one fraction of the venom only nor do they depend on the animal used for titration. The protective power measured by titration in rats is identical with that obtained by titration in the guinea-pig and is no higher against fractions of the venom prepared by the method described by Miss Freeman, Miss Williams and myself⁽²⁴⁾ than against the whole venom.

It may be admitted that the explanation of these phenomena might possibly depend on the inability of the antibody producing cells to put forth already formed antibody into the circulating blood, but no evidence is at present available in support of this hypothesis.

Direct Experiments on Cellular Immunity.

Natural immunity to poisons or toxins is seldom wholly due to the protective power of the serum. The natural immunity of the fowl and tortoise to abrin is not due to antitoxin in the blood (Calmette and Delarue⁽²⁵⁾) nor does the immunity of the hedgehog to eel serum depend on humoral immunity (Camus and Gley⁽²⁶⁾). The fowl is immune to tetanus toxin, yet its blood contains no antitoxin (Vaillard⁽²⁷⁾). The rat is highly resistant to diphtheria toxin, but its serum likewise contains no antitoxin (Pettit⁽²⁸⁾). It is usually assumed that the immunity is due to inability of the toxin to be fixed to the cells of the naturally immune animal, and certainly in the case of the fowl there is no antibody response to tetanus toxin, which may remain in the circulation for days without causing symptoms. Gunn⁽²⁹⁾ has shown that the natural immunity of the rat to strophanthus depends on cellular resistance, and Gunn and Heathcote,⁽²³⁾ comparing the resistance of the isolated perfused hearts of the cat and rabbit to cobra venom, showed that the resistance of the former animal is paralleled by the resistance of the perfused heart to the action of this venom.

In acquired immunity, apart from a single experiment by Gunn and Heathcote,⁽²³⁾ we are unaware that any clear cut results in favour of cellular as opposed to humoral immunity have been obtained, though numerous workers have addressed themselves to this problem.

In a single animal highly immune to cobra venom Gunn and Heathcote investigated the possibility of acquirement of immunity by the heart, by the smooth muscle of the intestine and by the red blood corpuscles. They found that in the normal rabbit heart perfused for twenty minutes to remove serum, cobra venom in a concentration of one in 200,000 caused systolic arrest in thirteen minutes, one in 400,000 in fifteen minutes, one in 600,000 in twenty minutes and one in 800,000 in thirty-four minutes. In the heart of a rabbit immunized to ten certainly lethal doses of venom and similarly perfused, one in 400,000 caused very slight effects in forty-five minutes and one in 200,000 of cobra venom caused systolic arrest in a further twenty minutes. This action of cobra venom in the normal rabbit is

attended by a diminution of the coronary flow from the first, whereas in the immune rabbit, as in the normal cat, there was a preliminary increase in the rate of flow.

They also showed that the effect of a concentration of one in 5,000 of cobra venom upon the isolated perfused intestine was conspicuously less in the immune rabbit than that produced by one in 10,000 in the normal rabbit. Finally, they found that the red blood corpuscles of the immune animal were more susceptible to hæmolysis by cobra venom than the red blood cells of normal rabbits.

But little attention has been paid to these important results, presumably because they depend upon a single unconfirmed experiment. It is not surprising that the red blood cells should prove to be sensitive rather than resistant, for, as Gunn points out, the red blood corpuscle can hardly be regarded as a reliable index of the presence or absence of cellular immunity. In natural immunity there is no evidence of any parallelism between resistance of the red blood cells and resistance of the whole animal and their transitory existence and the absence of a nucleus differentiate them sharply from other body cells.

In the experiments presented here we have avoided the influence of changes in the coronary flow, which might be offered as the explanation of the positive results obtained by Gunn and Heathcote, by using the perfused isolated rabbit auricle as our test object.

Our procedure has been to cut out the heart and perfuse it in the Locke Rosenheim apparatus, using the boric borate Ringer's solution described by Trevan as modified by Burn.⁽³⁰⁾ This has only half the amount of boric acid advised by Trevan and has a pH of 7.6. After 500 to 600 cubic centimetres of Ringer's solution have been perfused through the coronary circulation at a temperature of 34° C., the isolated auricle is put up in a bath holding 100 or 140 cubic centimetres of the same Ringer's solution and vigorously oxygenated. In some experiments the contractions of the auricle were recorded, but in many, to avoid the slight friction on the drum, observations were made of the auricle beating freely.

Twelve domestic rabbits were immunized against cobra venom, commencing on June 17, 1930, with a dose of 0.1 milligramme subcutaneously. They received increasing doses twice weekly, and by September 21, 1930, the dose was 0.88 milligramme. In half the animals immunization was then pushed more rapidly so that by October 30, 1930, they were receiving a dose of 3.9 milligrammes, while their fellows were receiving only 1.9 milligrammes. Some of the animals were now used for experiment a week after their last immunizing injection, but in the remainder immunization was continued until they could withstand a weekly dose of 9.5 milligrammes of venom. Two of these, numbers 11 and 12, were tested a fortnight and one (number 9) three weeks after their last immunizing injection.

The results of experiments upon the perfused isolated auricles of normal domestic rabbits, and of these immune animals are set out in Tables IV and V.

TABLE IV.

Effect of Cobra Venom on the Perfused Isolated Auricle of the Normal Rabbit.

Number of Observations.	Concentration of Venom.	Time in Minutes to Stoppage of Heart in Systole.
1	1 in 110,000	7
4	1 in 140,000	6, less than 10, 12½, 7
3	1 in 150,000	9, 17, 8
1	1 in 175,000	7½
5	1 in 200,000	13, 28, 29, 33, 23
3	1 in 220,000	68, 34, 105

Gunn and Heathcote's experimental findings are amply confirmed by these results. The normal auricle undergoes systolic contracture and ceases to beat in concentrations of venom of one in 110,000 to one in 150,000 in seven to seventeen minutes, and in concentrations of one in 200,000 and one in 220,000 in thirteen to thirty-three minutes and thirty-four to one hundred and five minutes. The immune auricles in these concentrations of venom do not undergo systolic contracture, but generally go on beating with a feeble flicker for prolonged periods.

By the use of this technique we excluded the possibility that the prolonged activity of the immune heart in Gunn's experiment was due to increased rate of flow in the coronary circulation of the immune, as opposed to a diminished rate of flow caused in the normal heart by cobra venom.

There remains for consideration the interpretation of these results. Can they be held to demonstrate an acquired tolerance of the cardiac muscle cells, which are specifically attacked by cobra venom, or do they only indicate the accumulation in or on the surface of the cells of antibody, which cannot readily be removed, even by prolonged perfusion? Further experiments incline us to this latter view. If the perfused auricle of a normal rabbit is suspended in the bath and even a relatively small amount of serum from an immune animal is added immediately before the addition of venom, there is delay in onset of systolic contracture and the auricle may even behave like one from an immune animal.

The serum of rabbit 9 was used for these experiments. This was titrated by hæmolysis tests, using

1.0 cubic centimetre of 5% suspension of guinea-pigs' corpuscles, 0.2 cubic centimetre of normal or immune rabbits' serum and amounts of venom varying from 1.0 to 0.01 milligramme. Normal rabbit serum has some protective effect, but the additional protective action of immune rabbit serum is such that 0.2 cubic centimetre of this appears to neutralize 0.05 milligramme of cobra venom.

Titration by protection experiments on guinea-pigs 1.0 cubic centimetre of serum 9 was found to neutralize 0.5 milligramme of cobra venom. When 0.2 cubic centimetre of this serum (an amount sufficient to neutralize at most only 0.1 milligramme of venom) was added to a bath of 140 cubic centimetres capacity in which a perfused auricle from a normal rabbit was suspended and 1.0 milligramme of cobra venom was subsequently added, the cessation times in three experiments were twelve and a half minutes, thirty-one minutes and one hour forty minutes. When half this amount of serum was used in three further experiments the observed times for cessation of beat were nine, twenty and forty minutes.

The addition of 0.2 cubic centimetre of normal rabbit serum to a bath containing a perfused auricle from a normal rabbit or the use of an unperfused auricle for the experiments gave cessation times which were within the limits of those given in Table IV. The serum of normal rabbits has no appreciable protective action against cobra venom in protecting guinea-pigs from the lethal action of the venom. It is clear, therefore, that the protection afforded by immune serum is due to a constituent which is not present in normal serum, that is, to antibody.

The protective effect of immune serum is very much greater when tested on the perfused rabbit auricle than is displayed in the protection experiments on guinea-pigs. So striking is this protective effect on the isolated rabbit auricle that these later results may even be held to raise the question as to whether the resistance observed in immune heart muscle may not actually be due to failure of perfusion to remove sufficient of the plasma from the auricle for its protective effect to be negligible. Possibly clotting takes place within the organ, not in the vessels, but in the tissue spaces, and sufficient antibody is held in this way to account for the phenomenon. The evidence obtained by the per-

TABLE V.

Effect of Cobra Venom on the Perfused Isolated Auricle in the Immune Rabbit.

Identification Number.	Weight of Animal in Kilograms.	Maximum Dose reached in Immunization in Milligrammes.	Concentration of Venom.	Time to Cessation of Beat.
8	2.63	1.9	1 in 140,000	Still beating feebly in 1½ hours.
18	2.32	3.6	1 in 140,000	Still beating feebly in ½ hour.
12	2.13	9.5	1 in 140,000	Cessation in 29 minutes.
11	2.1	9.5	1 in 140,000	Cessation in 1 hour 6 minutes.
13	2.5	3.6	1 in 150,000	Still beating feebly in 3 hours 20 minutes.
9	2.4	9.5	1 in 175,000	Cessation in 1 hour 20 minutes.
7	2.3	1.1	1 in 220,000	Still beating feebly in 65 minutes.
10	2.46	3.2	1 in 220,000	Still beating feebly in 2 hours 40 minutes.

fusion of organs must therefore remain suspect on these grounds and cannot be held to afford conclusive evidence of the acquirement of tissue immunity, either in the form of an acquired tolerance (independent of the production of humeral antibody) or of an accumulation of antibody in those tissues specially liable to attack.

The high active immunity of animals immunized with death adder venom, associated with extremely low titres of circulating antibody, remains to be explained, and the simplest available hypothesis is that antibody is removed from the circulating blood and accumulated in those tissues where it is most needed for defence against subsequent injections of the venom.

Conclusions.

We may sum up the conclusions arrived at as follows:

1. Active immunity to snake venom exhibits a greater degree of non-specificity than does passive immunity.
2. Its non-specificity depends on the similarity of the venoms in toxic constitution and on the close serological relations of the reptiles yielding them.
3. Closely allied venoms can act as specific secondary stimuli and cause the production of antibody, but it is doubtful whether the time required for this reaction is short enough to explain the high resistance of immune animals to venoms closely allied in toxic constitution.
4. The tissue immunity observed in perfusion experiments is probably determined by the accumulation of antibody in or on those tissue cells which are specially attacked by the venom. This cannot be removed even by prolonged perfusion.
5. This hypothetical accumulation of antibody in those tissues of the immune animal which are specially menaced by the poison used for immunization may account for the low titre of circulating antibody which is sometimes associated with a high degree of active immunity.

References.

- ⁽¹⁾ H. Noguchi: "Snake Venoms," 1909, page 233.
- ⁽²⁾ M. Phisalix: "Animaux Venimeux et Venins," 1922, Volume II, page 772.
- ⁽³⁾ C. J. Martin: "Curative Value of Calmette's Antivenomous Serum in the Treatment of Inoculations with the Poisons of Australian Snakes," *Intercolonial Medical Journal of Australasia*, August 20, 1897, April 20, 1898.
- ⁽⁴⁾ G. Lamb: "Specificity of Antivenomous Sera," Scientific Memoirs by Officers of the Medical and Sanitary Departments of the Government of India, Number 5, 1903, Number 10, 1904; "The Specificity of Antivenomous Sera with Special Reference to a Serum Prepared with the Venom of Daboia Russelli," Number 16, 1905.
- ⁽⁵⁾ S. Flexner and H. Noguchi: "Upon the Production and Properties of Anticrotalus Venin," *Journal of Medical Research*, 1904, Volume XI, page 363.
- ⁽⁶⁾ H. Noguchi: "Therapeutic Experiments with Anticrotalus and Antimocasin Sera," *Journal of Experimental Medicine*, 1906, Volume VIII, page 614.
- ⁽⁷⁾ F. Tidswell: "Researches on Australian Venoms," Department of Public Health, New South Wales, 1906, page 49.
- ⁽⁸⁾ M. Arthus: "De la spécificité des sérums antivenimeux, sérum anticobraïque et venins d'Hamadryas et de Krait," *Comptes Rendus de l'Académie des Sciences*, 1911, Volume CLIII, page 394. "De la spécificité des sérums antivenimeux, sérum anticobraïque, antithropique et anticrotalique, Venins de Lachesis lanceolatus, de Crotalus terrificus et de Crotalus adamanteus," *Idem*, page 1504.
- ⁽⁹⁾ B. A. Houssay: "Spécificité de l'action antitoxique des sérums antivenimeux," *Comptes Rendus des Séances de la Société de Biologie*, 1923, Volume LXXXIX, page 454.
- ⁽¹⁰⁾ V. Brazil: "La défense contre l'ophidisme," 1914, Second Edition, page 242.
- ⁽¹¹⁾ J. Vellard: "Spécificité des sérums anti-ophidiques," *Annales de l'Institut Pasteur*, 1930, Volume XLIV, page 148.
- ⁽¹²⁾ C. Phisalix: "Sur un nouveau caractère distinctif entre le venin de Vipéridés et celui des cobridés," *Comptes Rendus des Séances de la Société de Biologie*, 1904, Volume LVII, page 486.
- ⁽¹³⁾ C. H. Kellaway: "The Specificity of Active Immunity against Snake Venoms," *Journal of Pathology and Bacteriology*, 1930, Volume XXXIII, page 157.
- ⁽¹⁴⁾ C. H. Kellaway and F. Eleanor Williams: "The Serological and Blood Relationships of Some Common Australian Snakes," *The Australian Journal of Experimental Biology and Medical Science* (in the press).
- ⁽¹⁵⁾ G. H. F. Nuttall: "Blood Immunity and Blood Relationship," 1904, Section by G. S. Graham-Smith, page 336.
- ⁽¹⁶⁾ G. A. Boulenger: Catalogue of Snakes of the British Museum, 1896, pages 320-354.
- ⁽¹⁷⁾ N. Hamilton Fairley: "The Dentition and Biting Mechanism of Australian Snakes," *THE MEDICAL JOURNAL OF AUSTRALIA*, March 9, 1929, page 313.
- ⁽¹⁸⁾ A. T. Glenny: "The Principles of Immunity Applied to Protective Inoculation against Diphtheria," *Journal of Hygiene*, 1925, Volume XXIV, page 301.
- ⁽¹⁹⁾ A. T. Glenny and H. J. Südmerson: "Notes on the Production of Immunity to Diphtheria Toxin," *Journal of Hygiene*, 1921, Volume XX, page 176.
- ⁽²⁰⁾ H. R. Dean and R. A. Webb: "The Determination of the rate of Antibody (Precipitin) Production in Rabbit's Blood by the Method of 'Optimal Proportions'," *Journal of Pathology and Bacteriology*, 1928, Volume XXXI, page 89.
- ⁽²¹⁾ H. W. Acton and R. Knowles: "The Dose of Venom Given in Nature by a Cobra at a Single Bite," *Indian Journal of Medical Research*, 1913, Volume I, page 392.
- ⁽²²⁾ A. T. Glenny: "The Relation between Dosage and Death-Time," *Journal of Pathology and Bacteriology*, 1925, Volume XXVIII, page 251.
- ⁽²³⁾ J. A. Gunn and R. St. A. Heathcote: "Cellular Immunity: Observations on Natural and Acquired Immunity to Cobra Venom," *Proceedings of the Royal Society, Series B*, 1921, Volume XCII, page 81.
- ⁽²⁴⁾ C. H. Kellaway, M. Freeman and F. Eleanor Williams: "The Fractionation of Australian Snake Venoms. I. The Venom of the Death Adder (*Acanthophis antarcticus*)," *Australian Journal of Experimental Biology and Medical Science*, 1929, Volume VI, page 245.
- ⁽²⁵⁾ A. Calmette and A. Délarde: "Sur les toxines non microbiennes et le mécanisme de l'immunité par les sérums antitoxiques," *Annales de l'Institut Pasteur*, 1896, Volume X, page 675.
- ⁽²⁶⁾ L. Camus and E. Gley: "Sur le mécanisme de l'immunisation contre l'action globulicide du sérum d'anquille," *Comptes Rendus de l'Académie des Sciences*, 1898, Volume CXXVII, page 330.
- ⁽²⁷⁾ L. Vaillard: "Sur quelques points concernant l'immunité contre le tétanos," *Annales de l'Institut Pasteur*, 1892, Volume VI, page 224.
- ⁽²⁸⁾ A. Pettit: "Action de la toxine diphtérique sur le rat," *Annales de l'Institut Pasteur*, 1914, Volume XXVIII, page 663.
- ⁽²⁹⁾ J. A. Gunn: "The Congenital Tolerance of the Rat to Strophanthus," *Journal of Pharmacology and Experimental Therapeutics*, 1913, Volume IV, page 225.
- ⁽³⁰⁾ J. H. Burn: "Methods of Biological Assay," 1928, page 20.

THE VALUE OF CHOLECYSTOGRAPHY IN DIAGNOSIS.¹

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THE value of cholecystography as an aid in the diagnosis of gall bladder disease appears to be established, and the aim of this paper is rather to raise various points for discussion than to bring forward any fresh evidence on the subject.

In collecting data for this discussion, use has been made of the histories of patients suffering from gall bladder disease who have been admitted to the Mooroopna Hospital during the past three years. The incidence of these cases was first investigated. Statistics of the incidence of gall bladder disease in Australia were not available. In America, data from the Mayo Clinic⁽¹⁾ show that 5% of patients complained of gall bladder trouble. Nine per centum more had gross or microscopical evidence of disease of the gall bladder. Osler and McCrae⁽²⁾ state that cholelithiasis (cholecystitis is not mentioned) was found at autopsy in from 5% to 10% of subjects dead from all causes. Other figures⁽³⁾ quoted eighteen years ago are that gall stones are less frequent in the United States than in Germany, 6.94% to 12%, and that they are less common in England than on the Continent.

In investigating the number of patients diagnosed as suffering from gall bladder disease (acute cholecystitis, chronic cholecystitis and cholelithiasis) admitted to the Mooroopna Hospital during the past three years, this period was chosen for the reason that previously all the work at the hospital had been performed by a permanent medical superintendent and individual bias in diagnosis may have entered into the question. During the past three years, however, the patients have been investigated by any one of six honorary medical officers and various resident medical officers.

During the year 1927-1928, of all patients admitted over the age of fifteen years, 1.39%, and of those over the age of twenty-five years, 2.12%, were diagnosed as suffering from gall bladder disease. During 1928-1929 the numbers were 1.39% and 1.99% respectively, and during 1929-1930, 2.67% and 3.92%. The average incidence for the three years is 2.15% for the cases over fifteen years and 2.67% for those over twenty-five years.

An interesting point is the marked increase in the numbers diagnosed as suffering from gall bladder disease during the last of the three years, the figures being almost double those of each of the two preceding years. Whether this is due to a greater realization on the part of medical practitioners of a pathological gall bladder being the underlying cause of many indefinite cases of upper abdominal complaints or to the assistance of improved methods of investigation is impossible to determine.

The above figures do not reach those (5% to 10%) which are usually quoted as the incidence of gall bladder disease. The majority of the patients whose histories are available had marked or severe symptoms. There must be many more suffering

from mild symptoms due to gall bladder disease, who are not diagnosed as suffering from such, or whose symptoms are not sufficiently severe for further investigation to be undertaken at the time. In many of the cases under discussion the symptoms had been increasing in severity gradually, over a number of months or years, before the patients had submitted themselves for treatment.

The proportion of males to females has been found to be higher than the usual figure quoted, namely, 1 to 2. The figures are:

Year.	Males.	Females.
1927-1928	7	11
1928-1929	7	10
1929-1930	12	20

a proportion of about one to one and a half.

So that, although females are definitely more prone to gall bladder disease, the fact that a patient is a male does not place a diseased gall bladder among the rare conditions from which he might be suffering.

Classification According to Diagnosis.

In the diagnosis of disease of the gall bladder, the cases can be divided into two large classes:

1. Those with typical symptoms and signs of gall bladder disease in which the diagnosis is straightforward. These include (a) cases of acute cholecystitis, with evidence of an acute abdominal crisis, and (b) cases of cholelithiasis, with typical biliary colic.

2. Those cases in which the symptoms suggest some lesion in the upper half of the abdomen or in the right upper quadrant, but in which the symptoms are equivocal.

In the clinical diagnosis of cholelithiasis and chronic cholecystitis, apart from cases characterized by typical biliary colic, the usual syndrome is that of "flatulent dyspepsia" with pain of varying degree and localization, even confined to the left upper quadrant of the abdomen. In many of these cases the diagnosis is practically certain from the presence of any combination of the usual symptoms and signs of chronic cholecystitis, but in others there are occasional puzzling features and in which some further diagnostic procedure is felt to be necessary before the diagnosis is certain. It is in these that cholecystography should prove of value, taken in conjunction with the clinical picture.

Such cases are typified in one patient (Case III) whose symptoms were suggestive of gall bladder disease but the cholecystogram was that of a normal gall bladder and a barium meal showed visceroptosis.

A further example in another patient (Case XXX) who, in addition to the continuance of symptoms suggestive of chronic cholecystitis after a cholecystotomy had been performed, had also evidence of pelvic disease and whose upper abdominal symptoms were always worse at the time of menstruation.

Yet another patient (Case XXXII) was never seen in the attacks, which were on each occasion relieved after a bowel action; this patient was found by another practitioner to have tenderness in the appendiceal area after one attack.

These were personal cases.

A number of the patients had their fairly severe pain relieved after an evacuation of the bowels, and a large number complained of unusual constipation at the time of the attack.

¹ Paper read at a meeting of the Victorian Branch of the British Medical Association on October 11, 1930.

Cholecystography must always be considered as an aid to diagnosis, taken in conjunction with the patient's symptoms, and even if a pathological condition of the gall bladder is found in the cholecystogram, the possibility that the patient's symptoms may be due to some other independent lesion must not be overlooked.

Cholecystography is a method (i) of visualizing a normal or abnormal physiological change of the gall bladder and (ii) by the appearance of filling defects, of showing the presence of gall stones. A normally functioning gall bladder has the capacity of concentrating the bile contained in it. It therefore concentrates any dye present in the bile, and it is this concentration of dye, as shown in an increased density of the gall bladder shadow after an interval, that constitutes the diagnosis of a normal gall bladder. In chronic cholecystitis this function of concentrating bile is lost or impaired, and there is therefore no increase in the density of the second shadow. A further normal function is the emptying of the gall bladder after a meal, more completely and more rapidly after one containing fat, with a resultant complete or partial disappearance of the gall bladder shadow previously visualized.

Method Used.

Oral administration of the dye has been used throughout this series. There have been no ill effects through giving the dye by mouth except in a few of the earlier cases, when vomiting occurred. In two instances diarrhoea occurred after the patient took the dye. A second dose was given with no ill effects.

At first keratin-coated capsules of tetraiodophenolphthalein were given with sodium bicarbonate, the amount of dye administered being calculated on the body weight of the patient. In the later cases the powder has been given in half a tumblerful of water, the same amount—the contents of one bottle of "Shadocol"—being given to every patient irrespective of age, sex, or weight. No sodium bicarbonate is given. The dye is given in the evening following the usual tea meal and no further food is taken until after the second film is prepared. The first film is taken fifteen hours after the dye has been administered and the second film three hours later, that is, in eighteen hours. (In the earlier cases this was taken two hours after the first.) If the emptying capacity of the gall bladder is to be investigated (this is not done in all cases and, of course, not done if the first and second films show no shadow), a meal containing fat, usually bacon and eggs with bread and butter or cream, is then eaten and the third film taken two to three hours after the second, when in a normal case the shadow should have disappeared or nearly so.

The X Ray Technique.

X ray technique used is: 65 kilovolts for a patient of medium size, increased by two or three kilovolts for a fat person. The distance is 62.5 centimetres (twenty-five inches), the milliamperage is 100, and the time exposure is two seconds with the Potter-Bucky diaphragm; these are constant. Better

pictures with less fogging have been obtained by using the Potter-Bucky diaphragm.

Eighty-seven cholecystograms (eight patients have been examined more than once) have been done since July, 1927.

Analysis of Cases.

An analysis of the cases places them in the following groups:

Group I (a). Patients examined for outside medical practitioners, no history being recorded and no diagnosis made, twenty-five cases.

Group I (b). Films of no use through not being properly numbered, not being centred correctly, and definition bad on account of patients being very fat, six cases.

Group I (c). Gall bladder shadow not visualized, suggesting a pathological condition, but the dye present in the bowel, indicating non-absorption. This occurred in the earlier cases when keratin-coated capsules were used, two cases.

With correct or improved technique, Groups I (b) and I (c) should be eliminated, except the very fat patient, who will continue a problem. Group I comprises thirty-four cases.

Group II. There remain fifty-nine cholecystograms, constituting Group II, from which a diagnosis of normality or abnormality of the gall bladder and its ducts should be possible.

Group II (a). Sixteen patients in Group II were operated upon. In one case the cholecystogram was a poor one, so that fifteen cases remain for confirmation of the cholecystographic findings or not. The operative diagnosis of chronic cholecystitis requires to be considered here. Apart from the presence of stones and obvious thickening of the wall of the gall bladder, the diagnosis of a pathological gall bladder rests on the following findings: (i) Adhesions of the gall bladder to one or more adjacent organs. In association with the adhesions to the liver, scarring of the capsule of the superior surface of the liver in the vicinity of the chronically inflamed gall bladder is often seen. (ii) Abnormal appearance of the mucous membrane of the gall bladder. No mention is made in any of the cases of a "strawberry gall bladder," nor has there been one in the cases seen by the writer. In one, a few minute collections of a yellow crystalline substance, presumably cholesterol, were found close to the origin of the cystic duct. A condition occasionally seen is one in which areas of mucous membrane, pin head in size, are missing, being replaced by scar tissue. No microscopical examination of the removed gall bladder has been made. Apparently in order to obtain reliable evidence of pathological lesions, examination requires to be done immediately after removal, a condition which it is not possible to fulfil here. (iii) The so-called "biliary sand." This is taken to be an abnormal condition. (iv) Clinical improvement after operation with relief of symptoms complained of before operation. Immediate improvement cannot be relied upon. In several of the cases improvement after cholecystotomy lasted for a time, but the symptoms recurred later. In these

fifteen operative cases the cholecystogram findings were confirmed at operation.

Group II (b). In this group, comprising thirty-seven cases, the diagnosis of the condition was based on the clinical picture—history and physical examination. In thirty of these clinical diagnosis and cholecystographic findings agreed. In the remaining six the findings were not compatible.

In Case III the symptoms were very suggestive of gall bladder disease. The cholecystogram showed a normal gall bladder. The patient has not been seen since for confirmation or otherwise.

In Case V the history was lost. The condition was diagnosed as chronic cholecystitis, but the cholecystogram revealed a normal gall bladder.

In Case XV the history suggests gastric or duodenal ulcer with hemorrhage or subacute perforation. There is no concentration of dye in the second film.

This case raises the question as to whether it is necessary to have an increase in density of the shadow in the second film to diagnose a normally functioning gall bladder, or whether a shadow of definite but equal density in the first and second indicates a normal condition. Some authorities take the latter finding to indicate normality, but if concentration of bile is a criterion of normality, this process should continue until the time of taking the second film.

In Case XVII there was no shadow in five films. The patient was examined a second time, when the second film showed concentration with an irregular outline of the gall bladder and many filling defects.

In Case XXVI there was no shadow in the first film, but a definite small gall bladder in the second, it was empty in the third. The patient was examined a second time and the film showed a normal gall bladder. The patient had no definite symptoms of gall bladder disease. The result of the X ray examination was probably correct.

In Case XIV the history was defective. X ray examination records a pathological gall bladder. The patient probably has a gall bladder lesion.

The result of the investigation of these cases was interesting. In each of the fifteen cases operated upon (that is, those in which the cholecystogram was a good one), an abnormal gall bladder was shown to be present by the cholecystogram and a pathological condition was found at operation. Clinical evidence agreed with the cholecystogram findings in thirty out of thirty-seven cases, the remaining seven being inconclusive, as shown above.

In several patients, more than one cholecystographic examination was undertaken, but the final result was conclusive (apart from the above-mentioned seven cases) when good films were obtained.

In the cases in which the evidence was inconclusive, intravenous injection of the dye instead of oral administration would probably have clinched the matter.

Acknowledgements.

My thanks are due to the members of the Honorary Medical Staff of the Mooroopna Hospital for allowing me to utilize their clinical material, and to Dr. N. P. Wilson (Resident Medical Officer) and Mr. E. P. Doyle (Technician) for their assistance.

References.

- (1) Graham, Cole, Copher and Moore: "Diseases of Gall Bladder and Bile Ducts," 1928, page 110.
- (2) Osler and McCrae: "Modern Medicine," 1914, Volume III, page 569.
- (3) William Osler: "Principles and Practice of Medicine," 1912, page 569.

SWIFT'S DISEASE.¹

By C. H. DICKSON, M.B., B.S. (Melbourne),
Rushworth, Victoria.

I HAVE chosen for my subject Swift's disease for two reasons: firstly, I feel sure that many cases are undiagnosed on account of lack of familiarity with the clinical picture, and secondly, as a corollary, these cases are much commoner than is generally realized. In a twelve-month period in my own practice I have seen three undoubted cases, which I shall refer to later.

Thirty years ago this illness of children was recognized at the Children's Hospital by the late Dr. Snowball, Dr. Jeffreys Wood and others, but it was not until 1914 that Dr. Swift, of Adelaide, presented a paper on the subject at the Auckland Medical Congress and clarified the picture. Since then a large literature relating to the disease (under various names) has appeared, although the usual paediatric text books do not devote much space to it.

In regard to nomenclature, Dr. Swift used the term erythrædema, the Sydney physicians knew it as pink disease, British authors referred to it as a disease resembling *pellagra acrodynia*. There are objections to all these names, and Dr. Warthin, of the Pathological Laboratory of the University of Michigan, suggested the designation of Swift's disease, and that is the term by which it is becoming more generally known.

Distribution.

In the past ten years cases have been reported from practically every country in the world.

Ætiology.

The disease is essentially one of childhood. Dr. Jeffreys Wood has collected two series of cases, the youngest patient being an infant of five months, the oldest a child of three and a half years. The oldest cases have been reported from the American Continent in children of five years, but the greatest incidence occurs in the nine to eighteen months age group.

So far no causative factor has been recognized. Dr. Jeffreys Wood reports two cases in one family and a case occurring in a child one month after contact with a sufferer from the disease. Two of my own cases occurred in cousins residing in the same house, so the possibility of the disease being infectious cannot be disregarded.

The most exhaustive pathological investigation has failed to reveal any definite typical lesion.

The *post mortem* pathology is usually the pathology of complications.

¹ Read at a meeting of the Victorian Branch of the British Medical Association on October 11, 1930.

Dr. Reginald Webster, of the Children's Hospital, has made cultures from blood, fæces, and mesenteric glands without result.

Dr. Warthin, of the University of Michigan, sums up his *post mortem* findings as follows:

Acute infection of the respiratory tract was present in both cases, which also showed gastro-enteritis. Both showed marked evidence of the hypoblastic lymphatic constitution. In both cases there were extreme congestion and oedema of the central nervous system, with reticulo-endothelial proliferation. The essential pathological changes in these two cases would appear to be extreme oedema and slight meningeal irritation of the central nervous system, chronic erythema of the skin with hyperkeratosis, hypertrophy of the epidermis and sweat glands, with slight pigmentation of the rete, . . . with associated or terminal respiratory infections and gastro-intestinal catarrh and inanition. . . . The entire anatomical picture in these two cases suggests either a food deficiency or a toxin acting in persons of the hypoblastic constitution, affecting the reticulo-endothelial system of meninges and skin, the vegetative nervous system and possibly leading to a light sensitization.

He suggests that possibly the condition is an infantile variety of pellagra. Opposed to that theory we have the facts that while in this country pellagra is excessively rare, Swift's disease is not.

There is no particular seasonal incidence and no relationship between diet and onset of the disease has been detected. Sufferers may have been fed on breast milk, cow's milk, or any of the patent foods.

Symptoms and Signs.

Symptoms and signs may be summarized under ten headings: (i) Irritability, restlessness and sleeplessness, (ii) anorexia, (iii) sweating and sudaminal rash, (iv) wasting, (v) hypotonia, (vi) itching of skin with ulceration and infection, (vii) photophobia, (viii) stomatitis, (ix) redness and coldness of extremities, (x) complications.

I shall now consider the foregoing in detail.

Irritability, Restlessness and Sleeplessness.—Irritability, restlessness and sleeplessness are probably the most marked and troublesome symptoms of the group, and the earliest. Night and day these children cry, whine and toss in their cots, scratching at their extremities or rubbing them on the bedclothes or against each other. Frequently the hair is torn out by the roots.

Anorexia.—Anorexia is an early and well marked symptom, although if food can be forced on the child it is usually retained.

Sweating and Sudaminal Rash.—Sweating usually begins some two or three weeks after the onset of the illness. The sweats are frequently of a drenching character, but may be nothing more than a moistness of the skin. The sweats may at times cease for days and then recur in a more severe form. The rash is probably dependent on the sweating and comes and goes concurrently with the latter. It is miliarial in type with a pink tinge and is most pronounced on the back and front of the trunk, being especially marked under any constricting bands of clothing.

Wasting.—Wasting follows on the anorexia and restlessness and there may be a marked loss of weight. Dr. Wood quotes a patient losing 200 grammes (eight and one-third ounces) every week for eight weeks.

Hypotonia.—Hypotonia is noticed usually early in the illness. One of the first complaints of the mother is that the child "has stopped crawling" or, if old enough, "walking." The muscles show very marked lack of tone and it is possible to bend the limbs into all sorts of positions. It is this hypotonia, associated with the photophobia, which leads to the typical decubitus in these cases. In its cot the child just "flops," if I may use a colloquialism. The trunk is fully flexed with the head tucked away between the knees.

Itching of the Skin with Ulceration and Infection.—The itching appears to be, from the child's point of view, one of the most intolerable of the symptoms. The sufferers tear at their feet or hands with the nails, or rub them on the bedclothes until frequently the skin is broken and secondary infection ensues. I remember two patients at the Children's Hospital in 1927, one with extensive sloughing and ulceration of the dorsum of both feet, septic absorption from which was probably the ultimate cause of death, and another in which the lower lip had a semicircle about 2.5 centimetres (one inch) in diameter actually chewed out by the child.

Photophobia.—Photophobia although not invariably is very frequently present and gives a characteristic appearance. The child dreads the light and burrows its face into the bedclothes or the mother's chest. The eyes are half or completely closed and there is often a rapid and continuous blinking. The eyes externally do not show anything abnormal, except some conjunctivitis. Whether there are any fundal changes I am unable to say. I have found it impossible to examine the eyes with an ophthalmoscope on account of the restlessness and resentfulness of light, and it is unjustifiable to use general anaesthesia, as these patients are almost certainly bad "anaesthetic risks."

Stomatitis.—Stomatitis may be transient or severe. In the milder cases it usually clears up quickly, in others there is extensive ulceration of the mouth and gums with loss of all the teeth, which fall out spontaneously.

Redness and Coldness of the Hands and Feet.—Redness and coldness of the hands and feet may appear quite early in the disease or may be delayed for eight to twelve weeks after the onset. If present, they are pathognomonic and were responsible for the name "erythroedema." However, there is no true oedema, but the hands and feet are ice cold, swollen and red. A similar condition may appear on the face, thighs or buttocks. Desquamation usually follows and the nails may be shed.

Complications.—If death ensues, the usual cause is an intercurrent infection, especially of the respiratory or gastro-intestinal tract. Inanition and sepsis may also lead to a fatal termination. In other cases death occurs quite suddenly with no cause detectable *post mortem*. I have seen this occur twice in patients who were apparently recovering.

The above covers practically all the symptomatology with the addition that constipation is more common than diarrhoea and there may be fever which (in the

absence of complications) varies from normal to 37.8° C. (100° F.). Reflexes are present and often active, and so far as can be determined there are no definite sensory changes.

Case Reports.

The following cases are typical.

Case I.

S. T., a female, aged thirteen months, was first seen on February 9, 1928. She was the fourth child (the others being alive and well), was born at full term, and with the exception of "bronchitis with her first teeth" at four months had had no previous illness. The birth weight was unknown, but at three months she had weighed 7.6 kilograms (seventeen pounds). The present weight at thirteen months was 7.3 kilograms (sixteen pounds three ounces). She had been breast fed "at any time" and recently biscuits had been added to the diet. She began to crawl at twelve months. She had been perfectly well until one month earlier. She had then become irritable, had ceased crawling and refused her feedings. Since then she had cried night and day, buried her face in the pillow, evinced great objection to bright light, and had been scratching at her hands and feet. The mother volunteered the information that the child had suffered from profuse cold sweats and that the hands and feet were cold. Two months earlier a "blotchy" rash had appeared and persisted for a week. Afterwards the fingers had peeled and she now "had a rash which comes and goes." Bowels and micturition were normal.

Examination revealed a miserable "wingeing" child with typical blinking photophobia. There was a miliarial rash on the trunk, both back and front. Hands and feet were cold but not red nor swollen. There was no skin infection, and the mouth was healthy. There was marked hypotonia with hyperflexibility of the joints.

On February 20, 1928, the weight was 7.4 kilograms (sixteen pounds seven ounces), but the general condition was much as before.

On February 28, 1928, she had improved very much. There was no rash, but some sweating was still present. Irritability was less, but photophobia was pronounced.

On March 13, 1928 (two weeks later), weight was 8.2 kilograms (eighteen pounds two ounces). The child was very much better and trying to crawl again. Photophobia had diminished.

The child was not seen again until two years later when she developed pertussis, but otherwise was quite normal.

Case II.

Baby K. was first seen on July 21, 1928. He was aged nine months, the first child and there had been no other pregnancies. He was a cousin of S. T. (the previous patient), the mothers being sisters. The children had resided in the same house until two months previously.

The birth weight was unknown. At six months he had weighed 9.9 kilograms (twenty-two pounds), and when seen, 13.5 kilograms (thirty pounds). He had been breast fed since birth. He had always been a rather peevish baby, but for two weeks had been much more irritable than usual and for the past ten days had objected to the light. For one week he had been noticed to sit forward in his perambulator and to bury his head against the mother. He had had some transient rash on the trunk. For two weeks a cough had been present and at times had been followed by vomiting. The feedings were taken well. The bowels were normal. On examination he was a hugely fat child with blinking photophobia, irritable, and restless. There were scattered moist sounds in the lungs. The temperature was normal, and nothing else abnormal was detected. One week later his condition was unchanged. He was not seen again until September 17, 1928—six weeks later—when photophobia was very well marked and he was sitting in the typical position.

The mother had noticed that he was much flabbier than before. Examination then revealed a rash on the trunk, a mild generalized stomatitis, hypotonia and cold feet.

No further symptoms developed and a gradual recovery ensued.

This child was seen again a year later and was quite normal.

Case III.

Case III is not quite so typical, but I do not consider there is any doubt about the diagnosis.

Baby A., a female, aged one year and four months, was first seen on August 3, 1929. She was the first child and had been breast fed since birth. Since the age of ten months she had been irritable and sleepless and difficulty had been found in persuading her to take her meals. There had been repeated attacks of stomatitis in that period. No rash nor sweating had been noticed by the mother. For about four months she had resented the light and usually lay with her head buried in the pillow.

Examination showed a quite definite but not absolutely typical picture of the disease with photophobia, hypotonia, and irritability, but no rash, sweating nor changes on the extremities.

This child was ill for a further six months and developed pyelitis, which retarded her recovery.

Prognosis.

On the whole the prognosis is good in the absence of complications, but recovery is slow, the period of illness ranging from three to twelve months. The mortality rate is, however, far from negligible, especially among the public hospital type of patient. Of thirty-one patients admitted to the Children's Hospital in six years, ten died, but many of these were very ill on admission.

The possibility of sudden, unexplainable death must also be borne in mind.

Mortality is certainly greater in hospital than out of it, chiefly on account of the risk of cross infections in general wards.

Treatment.

There is nothing specific (the disease in any case is probably self-limited), but a good deal can be done to alleviate symptoms and prevent complications.

Keep the children at home and let them live in the open air night and day, avoiding exposure to the heat and glare of strong sunshine.

Clothing should be light and loose and any garments in contact with the skin should be of silk. Bedclothes must be as scanty as possible, as heat makes the skin irritability worse, and exposure of feet and hands to the air appears to afford some relief.

Swabbing the whole body twice daily with methylated spirits and painting the fingers and toes with 1% methylated tincture of iodine lessen the incidence of skin infections.

Charting of the arms to prevent scratching is also of value.

The stomatitis is best treated by swabbing the mouth with wool soaked in peroxide of hydrogen one part with four parts of water, followed by "Listerine," or allowing the child to chew wool soaked in peroxide of hydrogen.

Diet should be rich in vitamins and of full calorific value. If breast fed, do not wean the child. If not, whole raw milk should be given with fruit juices and "Ostelin" or cod liver oil.

For the sleeplessness an open air life gives most relief. Sedative drugs have proved of very little value. They are of more benefit if given to the mother, treatment of whom is also usually necessary, as the unfortunate woman is exhausted by the continual strain of trying to look after her child.

For the same reason admission of the child to hospital is at times necessary to give the mother rest.

Other methods of treatment have been used, especially the intramuscular injection of "Colossal calcium" for which good results have been claimed, and the use of ultra-violet rays, about which there is considerable difference of opinion. Dr. Ian Wood, Superintendent of the Children's Hospital, in a recent personal communication, told me that he does not consider that they influence the course of the disease in the slightest.

This morning I received a telephone message from Dr. Jean Macnamara. She asked me to express her regret for her inability to be present this evening and gave me the following information. It has been noticed recently that there is a possible relationship between poliomyelitis and Swift's disease. The character of the former disease appears to have changed in the past ten years and at times differentiation between the two illnesses has been difficult. Cases of both poliomyelitis and Swift's disease have occurred in the same street. Opinion is turning to the definite possibility of Swift's disease being either an aberrant form of poliomyelitis or due to a filterable virus closely related to the virus of infantile paralysis.

Dr. Burnet, of the Melbourne Hospital, is anxious to carry out investigations along these lines, following the work of Olitsky who discovered recently that if a monkey is infected with poliomyelitis, months after recovery of the animal cataphoresis of a cord extract results in deposition of virus at the anode and immune body at the cathode.

Dr. Burnet is anxious to obtain material for investigation and requests that if anyone comes across a child with Swift's disease, it should be sent to the Children's Hospital.

That concludes my remarks. There has been nothing original in my paper, but if it calls attention to a disease which is not uncommon and is not difficult to diagnose, I trust you will forgive me.

Acknowledgements.

In closing, I just wish to add that my sincere thanks are due to Dr. Jeffreys Wood who was kind enough to give me permission to plagiarize freely his articles which appeared in THE MEDICAL JOURNAL OF AUSTRALIA of February 19, 1921, and in THE MEDICAL JOURNAL OF AUSTRALIA (Supplement) of December 10, 1927.

Reports of Cases.

THREE CASES OF ECLAMPSIA.¹

By F. W. GRÜTZNER, M.B., B.S. (Melbourne),
Shepparton, Victoria.

It is not my intention to attempt a discussion of the very wide subject of eclampsia, but simply to present three of the cases which I have had and which are, I think, of peculiar interest in view of the after history of the patients.

¹Read at a meeting of the Victorian Branch of the British Medical Association on October 11, 1930.

Case I.

The first of the three patients in point of time is Mrs. G.A., aged twenty-five years, a *primipara*, who engaged me for her confinement in 1924 and who had her urine examined at moderately regular intervals. She ran a normal course in her pregnancy till February, 1925, when she began to show albuminuria. By February 15 this had become so marked that she was admitted to a nursing home where she remained for several weeks. Despite rest, dieting and free purgation, she showed no consistent improvement, but rather tended to get worse. Her systolic blood pressure, which ranged to 150 or 160 millimetres of mercury, was apparently much above its normal limit, as after her delivery it fell to 110 millimetres. As her condition caused much anxiety and she was almost by then at full term, in the second week in March, two attempts to induce labour were made by means of castor oil and quinine sulphate. There was no response whatever. On March 13 she had a series of fits, three or four in number. There was no sign of labour commencing and I decided on delivery by Caesarean section. This was duly done, a living female child resulting. The mother's post-operative course was fairly satisfactory. She had no more fits, her albuminuria steadily diminished and when she went home three weeks later the urine (despite an attack of cystitis at about the fifth day) was practically normal. She did not give me the opportunity to follow her up for any length of time, though I specially requested it.

I strongly advised against a second pregnancy, at any rate for several years, but in spite of advice and assistance, she did become pregnant in March, 1927. I was not privileged to attend her on this occasion and give now only her own statements in regard to it, but these bear the impress of truth.

In May or June, 1927, she saw a doctor in Melbourne about her forthcoming confinement. In November she went to her parents' home in the city. About this time her feet and legs began to swell very much, but she does not seem to have been ill till December 20, when she began to feel dizzy and her vision became blurred. She thought she was coming into labour and entered the nursing home for twenty-four hours. On December 23 she became completely blind and got very thirsty. At 11 p.m. the membranes ruptured and labour came to an end at 7 a.m. on December 24 with the birth of a living male child. From about midday, December 23, to midday, December 24, the patient was practically unconscious and knew of these details of labour only from her nurses. Her urine had not been examined till December 23 and was then "very bad."

She was in hospital three weeks after her confinement and says that her kidney condition and also her vision improved very quickly. There has been no further pregnancy and at the present time she seems very well.

Case II.

The second patient, Mrs. L.H., aged seventeen years, was a *primipara*. I first saw her at about 3 a.m. on May 28, 1928, on a bitterly cold night. She was then deeply comatose and, according to her husband, had awakened him with a cry and soon afterwards had a severe fit. She had two more very severe fits in my presence. She was just entering the ninth month of her pregnancy. Her feet and legs were grossly oedematous and indeed almost her whole body was swollen. Her urine was scanty, contained enough red blood cells to colour it and became solid on boiling. I gave her morphine 0.03 gramme (half a grain) to check further fits and removed her to a hospital. A slight dilatation of the cervix indicated the beginning of labour, but we proceeded with vigorous eliminative measures and the patient gradually regained consciousness after about twelve hours. She had had eight fits in all. Labour went on very satisfactorily. In the evening a living female child was born.

The urine cleared very rapidly, the convalescence was most satisfactory, and in about a month all apparent kidney trouble was over. I tested her urine very frequently for several months. The day after the child's birth the mother's blood pressure was 160 millimetres of mercury, but it soon fell to about 120 millimetres.

Unfortunately, to our surprise and dismay, she again became pregnant in March, 1929. I saw her every fourteen

days for the last seven months. On December 5 she did not seem very well, but was free of albumin and had no oedema. On December 18 she had a series of fits in the early hours of the morning. I had her removed to Mooroopna Hospital, where vigorous eliminative measures were adopted. Her blood pressure was 132 millimetres of mercury. She had no further fits and a few hours after admission induction of labour by means of the insertion of a rectal tube into the uterus was undertaken. On December 20 she gave birth to a still-born male child. Her subsequent course was on the whole satisfactory. Her urine quickly became free of albumin and on January 5, 1930, she was well enough to go home.

In April, 1930, a double salpingectomy was performed with a view to preventing further pregnancies. Her urine and blood pressure were quite normal whilst she was in hospital on this occasion.

Case III.

The third patient, Mrs. A.J.P., engaged me for her third confinement on December 14, 1929. I had attended her for the two other children, when she had quite normal pregnancies and confinements. The date of the third conception was very uncertain, but the due date was set down provisionally as June 21, 1930. On March 1, 1930, her urine was quite normal, but later on in the month contained a slight amount of albumin. A few days before April 19 she suddenly became oedematous and very distressed, but she did not consult me till the date mentioned. When I saw her, she was drowsy, looked very ill, had a systolic blood pressure of 182 millimetres of mercury and was generally oedematous. Her urine was solid on boiling. She came at once to Mooroopna Hospital. Eliminative measures were satisfactory and she rapidly passed out of the danger zone, but her urine still showed from "one-third" to "one-quarter" albumin. Her blood pressure stood at 150 to 160 millimetres of mercury. She was very anxious to have a living child and so it was decided to allow her to go on to the end of the eighth month and then to induce labour. Just at this time, however, Nature solved the problem and she was delivered naturally of a living child.

Her general condition improved with this, her blood pressure fell to about 110 millimetres of mercury and her urine cleared considerably, but in spite of rest in bed, dieting and so forth, always showed and has continued to show until the present a considerable deposit of albumin. There appears to be permanent renal impairment.

Comments.

Cases I and II are remarkable in that after apparent complete recovery from the toxæmia and its effects in the first pregnancy, the conditions were substantially reproduced for the second pregnancy. Probably in both cases it would have been wise to terminate the second pregnancy at the end of the eighth month.

Mrs. A.J.P. (Case III) was perhaps not technically an eclamptic, as she did not become comatose or have fits, but she was apparently on the verge of doing so, and therefore I feel justified in including her in this series. It now seems certain that it was unwise to have allowed the pregnancy to continue any longer than, say, one week after her admission to hospital, but, as already stated, a viable child was much desired. It seems quite clear that another pregnancy would be disastrous.

The three cases show how very difficult the management of such patients may be, and especially so as on the family physician devolves not only the treatment of their actual illnesses, but also the ordering of their lives. I trust that these imperfect notes may be of value to others in their dealings with a very important condition.

Reviews.

NURSES AND SURGERY.

A new edition of "Surgical Nursing," by Russell Howard, is a book which will be welcomed by nurses and all

engaged in the training of nurses.¹ The author who is an experienced teacher and examiner of nurses, has given in a concise form and from the nurse's point of view the principles upon which modern surgical treatment is founded, as well as the details of treatment.

The work is comprehensive, a surprising amount of instruction having been compressed into three hundred small pages. Little that could be included under the title has been omitted. The book is nevertheless easy and pleasant reading. The teaching is appropriately dogmatic. If the writer has his doubts and alternatives, he is wise enough not to confuse his student reader with them.

Of twenty-one chapters, seven are devoted to the nursing of special conditions, such as head injuries, rectal lesions, urinary diseases *et cetera*, and of these one chapter is devoted to ophthalmic nursing. It is surprising, therefore, to note that there is very little mention of the surgery of the nose and accessory sinuses and no special reference to gynaecological conditions.

The illustrations are not numerous (fifteen), but are well chosen. There is an appendix of useful formulae.

EDUCATION OF THE LAYMAN IN CANCER.

SEVERAL bodies in Australia devoted to the solution of the cancer problem have included amongst their declared activities the education of the public in the matter, but the task does not appear to have been undertaken very seriously so far. In "Cancer and Scientific Research" Barbara Holmes, Ph.D., of the Biochemical Laboratory, Cambridge, writes for the information of the lay public.² In a preface the President of the Royal Society emphasizes the importance of interesting the layman in the causation of cancer rather than in its cure, believing that an interest so created would tend to dissipate hopeless and superstitious feelings. The little volume is well written in good English, directly expressed, and is divided into short chapters, the distinct subject matter of each being concisely arranged and complete; where explanations are necessary, they are made with great care and in non-technical language; therefore, those for whom the book is intended should find it easy reading. The printing is good and the type large.

The history of the cancer problem makes a very interesting chapter. Statistics are explained and attention drawn to the discrimination needed for their interpretation and for the elimination of fallacies. The influence of irritation is clearly indicated in the chapter on occupational cancers, and the reader is made familiar with the methods and valuable results of animal experimentation. Succeeding chapters on inheritance, transplantable and transmissible tumours, immunity and tissue culture introduce the layman to many unexpected phases of the research and indicate to him the wide issues involved.

Dr. Holmes is not concerned with describing the minute anatomy of the tissues and explaining the physiological processes underlying normal growth and the departure in cancer from those processes, which is so necessary if the layman is to appreciate the significance of cancer development and the mechanism of metastasis; nor does she deal with the naked-eye appearance of cancer or with its early diagnosis and treatment. The authoress adheres strictly to the object of the book, which is to bring before the lay public the methods and results of scientific cancer research, particularly in its experimental and statistical aspects.

In any scheme for instructing the lay public in the cancer problem the diffusion of appropriate literature must play a part, and therein this book would fill an important place. It will prove of great interest and value to a selected class of readers and be very welcome to many practitioners.

¹ "Surgical Nursing and the Principles of Surgery for Nurses," by Russell Howard, C.B.E., M.S., F.R.C.S.; Sixth Edition; 1930. London: Edward Arnold and Company. Crown 8vo., pp. 352, with illustrations. Price: 7s. 6d. net.

² "Cancer and Scientific Research," by B. Holmes, Ph.D., with a preface by F. G. Hopkins; 1931. London: The Sheldon Press. Crown 8vo., pp. 168. Price: 3s. 6d. net.

The Medical Journal of Australia

SATURDAY, JULY 4, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

HIGHER DEGREES IN SURGERY.

By conferring a degree on a successful student a university shows that the student has satisfied certain requirements and has given proof of his knowledge to his examiners. Originally the title of master or doctor was nothing more than a licence to teach. The degree of master was first conferred by universities of France and Italy in the twelfth century. Later on the degree of doctor largely replaced that of master. In the process of time British universities have adopted the three grades, bachelor, master and doctor. It was natural that universities in so young a country as Australia should follow the British custom of granting a degree at graduation and a higher degree after a certain effluxion of time to indicate the acquisition of special knowledge or the possession of particular ability in a certain direction. In medicine the degree obtained at graduation is one qualifying the holder to be registered as a medical practitioner. Unfortunately, the three Australian universities did not originally adopt either the same standards or the same designations for their degrees. In each university the qualifying degree was that of bachelor of medicine (M.B.). The Universities of

Melbourne and Adelaide also granted the degree of bachelor of surgery (B.S.) to graduands. In Sydney a newly qualified graduate, a bachelor of medicine, was admitted on payment of £10 to the degree of master of surgery (Ch.M.); there was no bachelor of surgery degree. Melbourne and Adelaide required graduates to satisfy examiners either by thesis and examination or by thesis alone before they were granted the degree of master of surgery (M.S.). The position was absurd. It is true that Sydney graduates could take their doctorate of medicine in surgery, but this did not alter the absurdity. As far as can be discovered, the first move towards uniformity was the passing of a resolution by the Section of Surgery at the Eleventh Session of the Australasian Medical Congress at Brisbane in 1920, as follows:

That the members of the Surgical Section of the Eleventh Australasian Medical Congress are of the opinion that an endeavour should be made to secure, as far as possible, uniformity in the surgical degrees granted by the several universities of Australasia.

Several years after the passing of this resolution the University of Sydney abolished the degree of master of surgery (Ch.M.) and substituted that of bachelor of surgery (B.S.). It instituted a new degree, the master of surgery (M.S.). This degree has never been awarded. An important step towards uniformity was thus taken, but the conditions governing the degrees at the three universities were still widely different.

The next step took place at the instigation of the Royal Australasian College of Surgeons (or, as it was then, the College of Surgeons of Australasia). A conference of the Deans of the Universities of Sydney, Melbourne and Adelaide was held in March, 1929. Sir George Syme, the President of the College, was present by invitation. Certain recommendations were made at this meeting. They were discussed at a further conference in March, 1930. Approval was given to uniform regulations governing admission to the M.S. degree in general surgery, and it was suggested that there should be special diplomas or degrees in the various specialties. The recommendations of the conference were submitted to the three universities. At present no further reference will be made to diplomas or

degrees in special subjects such as ophthalmology, gynaecology *et cetera*. They will be discussed on a subsequent occasion. As far as the M.S. degree in general surgery is concerned, uniformity has been reached with one exception. In all three universities the examination will consist of two parts. In Melbourne and Adelaide Part I will consist of anatomy and physiology (including biological chemistry). In Sydney Part I is similar except that pathology is added. Part II will consist of an examination in general surgery which may or may not include research. In Melbourne and Adelaide pathology will, of course, be included in Part II.

The degree of uniformity already attained is most satisfactory and reflects credit on the authorities of the three universities. There would be no urgent need to ask that the uniformity be made complete were it not for another factor. Arrangements have recently been made for the primary examination for the Fellowship of the Royal College of Surgeons of England to be held in Australia. It has been suggested that the passing of the primary fellowship examination should be recognized by the Australian universities as being equivalent to the passing of Part I of the examination for the M.S. degree in Australia. The University of Melbourne has adopted a regulation to this effect. The University of Sydney will be approached by the Royal Australasian College of Surgeons with the request that pathology be transferred from Part I of the examination for the M.S. degree to Part II. The Royal Australasian College of Surgeons has no axe to grind in the matter, beyond the advancement of the art and science of surgery. It is to be hoped that the Senate of the University of Sydney will not turn a deaf ear. We would suggest that the examinee should be given as much consideration as possible. If the same result can be obtained by confining Part I of the M.S. degree examination to anatomy and physiology, and if the path of the examinee can be made a little less thorny, Sydney should fall into line with Melbourne and Adelaide. Sydney and Adelaide might then consider the possibility of accepting the primary fellowship examination as equivalent to Part I of the examination for the degree of master of surgery.

Current Comment.

A NEW RENAL EFFICIENCY TEST.

CONSIDERABLE interest has been shown recently in the article by D. D. Van Slyke and his coworkers on Bright's disease in *Medicine*, September, 1930, not only in the classification of kidney disease there set forth, but also in the one renal efficiency test to which the authors pin their faith. This is known as the blood urea clearance. Van Slyke, McIntosh, Möller, Hannon and Johnston found in 1930 that the relative sensitiveness to renal damage of this clearance was such that it indicated a drop below 40% of normal function in a large percentage of cases of progressing nephritis when phenolsulphonephthalein excretion, blood creatinine and blood urea, considered without its relation to urea excretion, were still within the range of normal variation, and that, in point of time, the blood urea clearance in progressing nephritis revealed development of functional deficit weeks or months before the other tests above mentioned.

The theory and technique of the test have been set out by E. Möller, J. F. McIntosh and D. D. Van Slyke,¹ and, as this is not generally available, it has been thought that the following brief *résumé* may be of interest.

Marshall and Davis and others have shown that when the urine volume is fairly large the rate of urea excretion is directly proportional to the blood urea content. Austin, Stillman and Van Slyke have shown that this direct ratio only holds when the urine volume is above a given limit—about two cubic centimetres per minute in adults. Below this, which they term the augmentation limit, the urea excretion rate falls in proportion to the square root of the urine volume. The amount of urea excreted per minute over the augmentation limit is known as the maximum blood urea clearance or C_m . Expressed as an equation:

$$(1) \quad C_m = \frac{U \times V}{B}$$

where U equals the urea concentration in urine, B the urea concentration in blood, and V the volume of urine excreted per minute.

Below the augmentation limit the volume of blood, the urea content of which is represented in one minute's excretion (the blood urea clearance per minute), is not a constant, but varies on the average in proportion to the square root of the urine volume. In order to compare excretions below the augmentation limit, therefore, they must be observed either with a standard, constant urine volume output (which is practically impossible) or, if observed with other urine volumes, the excretion rates must be corrected. For the excretion measured with any volume below the augmentation limit the following formula has been arrived at. If C is the

¹ *Journal of Clinical Investigation*, December, 1928.

observed blood urea clearance with any urine output, V , below the augmentation limit, then with the standard urine volume, V_s , the corresponding standard clearance, C_s , may be calculated by the square root rule:

$$C_s : C = \sqrt{V_s} : \sqrt{V}$$

$$\text{or } C_s = C \sqrt{\frac{V_s}{V}}$$

The standard urine volume adopted is one cubic centimetre per minute—the average rate of normal excretion per minute for adults (1,440 cubic centimetres per twenty-four hours)—and it has the advantage of simplifying calculation. We therefore have:

$$C_s = C + \sqrt{\frac{1}{V}}$$

or substituting for C :

$$C_s = \frac{U \times V}{B} \times \sqrt{\frac{1}{V}}$$

$$(2) \text{ that is: } C_s = \frac{U}{B} \times \sqrt{V}$$

the standard clearance, or the efficiency with which the kidneys excrete urea when the urine volume is at the average normal level of one cubic centimetre per minute. The maximum clearance indicates the maximum efficiency of urea excretion with high urine volumes and is normally about 40% greater than the standard clearance, or 75 cubic centimetres and 54 cubic centimetres of blood per minute respectively. (Note: Any convenient units of urea or urea N concentration may be used to express U and B , so long as the same unit is used for both U and B . The unit for expressing V cannot be changed without changing the numerical values of C_s or C_m .)

McIntosh, Möller and Van Slyke (1928) showed that calculated blood urea clearances may be corrected for variations in body size by a factor, based on the assumption of Addis that excretion varies directly as surface area. The corrected volume in such cases, V_{cor} , is the observed volume per minute, $V \times \frac{1.73}{A}$, where A is the body area in square metres normal for the subject's height and age. In the standard clearance in persons between 62 and 71 inches in height correction for body size does not exceed 5% and may be neglected; in the maximum clearance the range of height with less than 5% correction is 65 to 69 inches. To determine A in adults over 25 years the patient's height and the surface area for 25 years of age are used.

The procedure is as follows: If the urine volume exceeds two cubic centimetres per minute as observed in an adult, or as corrected for body size in a child, the maximum clearance is calculated. If the volume thus observed or corrected is less than two cubic centimetres per minute the standard clearance is calculated.

It is advantageous to calculate both in percentages of the mean normal C_s and C_m , thus expressing directly percentages of average normal renal efficiency. The percentage of average normal C_m is obtained by dividing the absolute C_m value by 75 and multiplying by 100 or, briefly, by multiplying by the factor 1.33; and the percentage of average normal C_s by multiplying by 1.85 ($\frac{1.90}{54}$).

Möller, McIntosh and Van Slyke adopt the following technique: No previous routine is adopted, except that vigorous exercise is avoided and the previous meal should be a moderate one (preferably without coffee, which Addis and Drury (1923) have found may increase the blood urea clearance). The test is usually made in the hours between breakfast and luncheon, when excretion, according to Mackay (1928), is least liable to fluctuation. The patient remains quiet while the urine is collected during two succeeding periods of one hour each. The chief source of error is probably the possibility of incomplete emptying of the bladder either at the beginning or end of a period. The collection of two specimens affords a check on this factor. A few minutes before the end of the first hour a blood sample is drawn. Its urea content is used for calculation of the clearance during both periods. This is permissible because under the test conditions the blood urea does not change greatly during an hour. Two examples may be given:

Calculation of Maximum Clearance.

Blood urea N = 15.6 mgm. per 100 c.cm. = B .
Urine urea N = 321.0 mgm. per 100 c.cm. = U .
Urine volume = 210.0 c.cm. per hour.
= 3.5 c.cm. per minute = V .

$$(1) C_m = \frac{U \times V}{B} = \frac{321 \times 3.5}{15.6} = 72 \text{ cubic centimetres of blood cleared of urea per minute.}$$

Percentage of average normal function = $72 \times 1.33 = 96$.

Calculation of Standard Clearance.

Blood urea N = 14.7 mgm. per 100 c.cm. = B .
Urine urea N = 750.0 mgm. per 100 c.cm. = U .
Urine volume = 50.0 c.cm. per hour.
= 0.83 c.cm. per minute = V .

$$(2) C_s = \frac{U \times \sqrt{V}}{B} = \frac{750 \times 0.91}{14.7} = 46 \text{ cubic centimetres of blood cleared of urea per minute.}$$

Percentage of average normal function = $46 \times 1.85 = 85$.

Decrease in the volume of blood cleared of urea per minute in pathological conditions must be due to one of two causes: either the volume of blood per minute passing through the kidneys is diminished (as is probable in cardiac decompensation and glomerular nephritis) or the proportion of its urea removed during that passage is less than normal (a possibility on which the authors do not yet express an opinion). They give the probable variation of the standard blood urea clearance as $\pm 10\%$, and point out that there are undoubtedly other factors affecting urea excretion besides blood urea concentration and urine volume.

Abstracts from Current Medical Literature.

RADIOLOGY.

Sacro-Iliac Slip.

W. EDWARD CHAMBERLAIN (*The American Journal of Roentgenology*, December, 1930) describes a method of studying sacro-iliac slip. In studying the pelvis, the entire pelvic brim must be studied stereoscopically and in addition the following films taken: A lateral projection centred at the lumbosacral junction and two projections of the symphysis pubis in which the patient is standing facing a vertical Potter-Bucky diaphragm, one film with the patient's weight borne on the right leg and the other with his weight borne on the left leg. The latter two films, when they show demonstrable alteration of the level of one pubic bone with relation to the other, are taken to indicate innominate bone rotation and sacro-iliac relaxation.

Tuberculosis of Intervertebral Articulations.

HOWARD P. DOUB AND CARL E. BADGLEY (*American Journal of Roentgenology and Radium Therapy*, March, 1931) describe tuberculosis of the intervertebral articulations. The classical type of Pott's disease is readily diagnosed, when the process is extensive enough to produce obvious clinical signs with collapse of the vertebrae, kyphosis or the pointing of a cold abscess. Earlier recognition, however, is essential to obtain a chance of cure without deformity. Two types of the disease are recognized early; those with central involvement of the body and early bone destruction of the anterior half of the body. A third type is frequently missed until late. This has been called the epiphyseal type, and it is this type which is the great source of failure of early recognition. The authors prefer the term "intervertebral articular tuberculosis" to that of "epiphyseal tuberculosis of the vertebral body." In discussing the pathology they state that tuberculosis of the spine is a local manifestation of a generalized tuberculous infection. The spread is by the hæmatogenous route, though the lymphogenous route may play a part. The anatomical position of the infection would therefore be dependent on the blood supply to the individual vertebra, the details of which are described by the authors. Various areas are involved, depending on the distribution of the blood vessels carrying the infection. They are: (i) The central type with collapse of the body and kyphosis; (ii) the epiphyseal type in which there is early narrowing and disappearance of the disc, the first clinical symptom is abscess formation, whilst collapse and kyphosis are late; (iii) the anterior variety with infection beneath the anterior longitudinal ligament; (iv) more rarely the vertebral appendages may

be involved. In the central form, as well as in the anterior type, the normal formation of the bone cannot be retained and kyphosis occurs. In the intervertebral type, however, the vertebral body is preserved intact until late in the disease. Of Röntgen signs the first is a narrowing of the intervertebral space, which may become quite marked before there are noticeable changes in the vertebrae. The normal size and shape of the body are preserved until the late stages. The authors quote three cases of the intervertebral articular type, in which abscess formation was the first intimation of the true nature of the disease.

Radiography with "Skiodan."

H. C. OCHSNER AND W. N. WISHARD, JUNIOR (*American Journal of Roentgenology and Radium Therapy*, March, 1931) give a preliminary report on urinary tract Röntgenography by means of "Skiodan." The authors describe the history of the researches which led to the production of pyelograms, following intravenous injections, until the introduction of "Uroselectan," the use of which has stimulated additional work. "Abrodil," which is the same as "Skiodan," was introduced in April, 1930, and is the sodium salt of iodomethane sulphonic acid. It is a stable compound, absolutely unchanged by boiling, sufficient for sterilization. The iodine content is 52%. The chemical composition is such that it is not attacked in metabolic processes. The theoretic maximum dose for a 60 kilogram man is 120 grammes. In clinical studies, when 40 grammes were used, occasional symptoms were noticed. Twenty grammes administered intravenously in one hundred cubic centimetres of distilled water produced an equally good pyelogram and no symptoms. The local effect on the veins is slight, and small extravasations into the subcutaneous tissues produced burning pain for about ten minutes, but there was no further effect. The solution of twenty grammes in one hundred cubic centimetres of doubly distilled water is sterilized by boiling and should be injected at body temperature. The injection should be as rapid as possible, for rapid administration produces no ill effects and excretion begins almost immediately. The first film should be taken five minutes and the second fifteen minutes after injection. In almost every instance in which injection of one hundred cubic centimetres of solution has been accomplished without mishap, in four to eight minutes good visualization of the entire urinary tract has been obtained. In the absence of obstruction the density of the shadow is never so great as that obtained in retrograde pyelography. The authors are against the use of compression in taking the skiagrams in view of possible interference with function, which is part of the information to be obtained. They have met with no success in the rectal administration of the drug. The oral administration

is also unsatisfactory, owing to slow absorption and obscuring of the kidney shadows by the unabsorbed medium in the bowel. The indications for intravenous pyelography are quoted from von Lichtenberg and Swick. Intravenous pyelography is indicated: (i) when cystoscopy and ureteral catheterization cannot be performed, (ii) because of obstruction in the ureter with blockage of the retrograde pyelogram, (iii) when direct pyelography cannot be undertaken. Intravenous pyelography is not intended to supplant the older direct method.

Radioscopy and Radiography in the Gastro-Intestinal Tract.

RENE A. GUTMAN (*La Presse Médicale*, February 14, 1931) describes the respective indications for radioscopy and radiography in lesions of the gastro-intestinal tract. The author states that there is a prevailing opinion amongst medical men and sometimes even among specialists that the important technique in gastro-intestinal examination is radioscopy and that a good screen examination is sufficient to establish diagnosis in most cases. Every year a certain number of medical practitioners, in spite of the lessons they have received, instal an apparatus sufficient for radioscopy and imagine that they will find in this manner light upon their diverse cases. That they thus commence a career pregnant with diagnostic errors. Duodenal ulcers, the most frequent of all organic lesions, escape almost altogether the screen examination. This is not a reflection on the competence or the knowledge of the observers; the author has several times, after a diagnosis immediately and easily made from films of duodenal ulceration, tried in vain to find the deformity on the screen. It is true that some of the lesions are so gross and so fixed that even the screen discloses them, but, generally speaking, apart from an irregularity of form, nothing is seen at the screen examination, and this sign is nothing but the mark of irritation of the bulb, from whatever cause, whether ulcer, gall bladder or appendix, and although duodenal ulcer is the most frequent cause of the deformity, its presence indicates a probability and not a certainty of diagnosis. These remarks apply to the classical ulcer deformity, but the ulcer niche may be the only sign visible on a bulb whose contour appears absolutely intact, and this niche is almost never seen on the screen. In juxta-pyloric ulcers the author states that on the screen there are more chances of allowing the lesion to pass undiagnosed or to be diagnosed unduly, than when good plates are taken. Ulcers of the lesser curvature are often so gross that advocates of radioscopy alone are confirmed in the idea that they see all ulcers and that when they do not see them there are none. Unhappily, he states, this is not the case, and he has collections of plates showing little

niches quite invisible on the screen. In pyloric stenosis both radioscopy and radiographic methods offer little difficulty in diagnosis when well established. The author does not question the utility of radioscopy examinations in conjunction with radiographic. It gives an idea of the fixity or otherwise of a lesion and the stiffness of the stomach wall; the peristalsis and gastric evacuation can also be studied, but a constant insistence on the necessity for the taking of serial films is a feature of the article.

PHYSICAL THERAPY.

X Ray Treatment of Cerebral Fistulae.

M. SGALITZER (*Wiener Medizinische Wochenschrift*, September 6, 1930) describes the treatment of cerebral fistulae by the use of X rays. The majority of these fistulae follow operations for cerebral tumour, others are post-traumatic and in rarer instances the cerebro-spinal fluid escapes through the cribriform plate, due to increased tension from cranial tumours or from some unknown source. The author employed four fields, ten by twelve centimetres, over the forehead, both parietal regions and the occiput. The focal distance was thirty centimetres and a dose approximately 50% to 60% of the minimum skin erythema dose was given. This was repeated in four to eight days. The details of nine cases are given; in all the fistula was soon healed.

Bone Metastases.

MURRAY M. COPELAND (*Radiology*, February, 1931) describes malignant lesions localized in bones, together with the pathological processes involved and the treatment. In regard to the breast, the treatment of the osseous lesions was considered for purposes of analysis on three groups of patients: first, those who had a radical amputation of the breast; second, those who had only simple breast amputation or local excision; and third, those on whom no operation was performed. The X ray treatment consists of twelve thirty-minute exposures, 200 kilovolts and five milliamperes being used; the rays were filtered through 0.075 millimetre of copper and one millimetre aluminium, with a 25 centimetre diaphragm opening and a 50 centimetre focal skin distance. A total exposure of ninety minutes is given over each portal of entry in doses of thirty minutes each on consecutive days, four portals of entry being used. In Group I two patients out of 74 survived the metastases for 71 and 48 months respectively. After irradiation by X rays the average duration of life was 18 months, as compared with 11.5 months for those who received no X ray therapy. In Group II the average duration of life of those patients with metastases who received X ray therapy, was 16.2 months, as compared

with 12.8 months for those who were not so treated. In Group III the patients irradiated by X rays lived ten months, while those without X ray therapy lived 7.8 months. Resection of the affected part apparently had no effect on the duration of life, but did relieve pain, while X ray therapy gave relief from the excruciating pain experienced in the diseased bone and in some instances definitely prolonged life. In regard to the prostate, upon gross examination of the material on hand, the metastatic nodules appeared as white or greyish nodules, surrounded and often permeated by a healing bone reaction. This response to tumour invasion was found to be quite the reverse of that usually seen in other metastatic lesions, except where osteoclastic lesions had been treated by X ray therapy. This bone reaction to prostatic carcinoma suggested strongly the hypothesis that the invasive powers of the secondary tumour deposits were of such moderate character that bone proliferation kept pace with the tumour invasion. Röntgen therapy offered relief from pain, but was not effective in eradicating the lesion or in greatly prolonging life. In regard to the uterus, in metastatic bone lesions Röntgen rays and radium proved particularly unsatisfactory. Not a few of the patients with melanoma were relieved from pain for long periods of time by X ray therapy, and in a few instances they lived from three to eight and a half years following X ray therapy or amputation of the affected part. In metastases of bone from primary hypernephroma, primary malignant disease of the testicle, bladder, ovary, thyroid and gastrointestinal tract, the author found that X ray therapy relieved pain, but that no permanent improvement resulted.

Lesions of Bone.

CHARLES F. GESCHICKTER (*Radiology*, February, 1931) sets out thirty-one rules for the diagnosis and treatment of bone lesions. He states if acute osteomyelitis is excluded, X ray films should be used for consultation when doubt exists before surgical operation is undertaken. Surgical operation is rarely indicated in multiple lesions and in the minority of single lesions. There is no harm in waiting for a confirming opinion. While the surgeon is waiting the part should be put at rest and deep X ray or radium therapy should be given to determine radiosensitivity. Four out of the eight types of solitary bone tumour are radiosensitive; this provides a therapeutic test. Tumours of the small bones of the hands and feet (excluding the *os calcis*) are usually benign. A central tumour of the sternum is a benign chondroma. A central bone-destructive lesion in the epiphysis of the lower end of the radius which expands the cortex, is practically always a giant-cell tumour. Diffuse bending or bowing of the bone is in favour of a benign lesion. Mul-

tiples lesions in children and in adolescents up to twenty years of age are benign, excepting bone dissemination from malignant disease in internal organs and in late Ewing's tumour. Multiple lesions in adults are malignant, except Paget's *osteitis deformans*, osteomalacia and distinct joint lesions. Lesions characterized by periosteal bone formation in adults are usually benign, whereas in patients under twenty this usually indicates malignant disease, unless the bone formation surmounts a pedicle of normal bone. In patients over thirty bone-destructive lesions that escape beyond the cortex, are usually malignant, unless occurring in an epiphysis in the small bones of the hand or foot, or at a joint. Deep X ray therapy should be discontinued in favour of surgical operation if definite results are not obtained in six weeks. A sarcoma at the upper end of the femur has never been cured by amputation. In the bones of the arm and the fibula a radical resection offers as much for the cure of malignant disease as amputation. The following tumours of bone are radiosensitive: Ewing's sarcoma, chondral forms of osteogenic sarcoma, highly cellular forms of fibrosarcoma, giant-cell tumour and giant-cell variants of bone cysts. Pain is relieved and life lengthened by irradiation in metastatic carcinoma. Deep X ray therapy may be used to control pain in bone disease temporarily, except in cases in which there is an acute infection.

Deep Röntgen Ray Therapy of Mammary Carcinoma.

WILLIAM A. EVANS AND T. LEUCUTIA (*American Journal of Roentgenology and Radium Therapy*, December, 1930) give a detailed statistical account of the "five year results" obtained in a series of 173 cases treated between 1922 and 1924. All patients, with the exception of nine, were followed up. They summarize these results as follows: Röntgen therapy, in its short wave length application, is of considerable aid in treatment of operable mammary carcinoma as an auxiliary to surgical procedures. Whereas in the cases without glandular involvement the addition of irradiation does not seem to increase noticeably, the five year results above those of surgery alone, assuming, of course, that all the diseased tissues were removed, in the carcinomata which had already spread to the axillary or the high thoracic lymph glands, the five year "cures" are nearly doubled. The best method of procedure is a combination of radical surgical operation with a thorough systematic irradiation of the entire anterior part of the thorax, axilla and neck on the diseased side. In the inoperable or recurrent mammary carcinomata Röntgen therapy produces prolongation of life if the lesion is localized to the adjacent lymph glands or anterior part of the thorax, and alleviates the symptoms if the carcinoma is already generalized.

Special Article.

INCOME TAX: FEDERAL AND STATE.

THE financial year having come to a close on June 30, 1931, it now becomes necessary for all members of the medical profession to prepare a return of their income for that period. Except in certain circumstances (referred to later), it is necessary for each taxpayer to prepare only one combined Federal and State return, which will be used by both departments in assessing the amount taxable for Federal and State purposes.

No doubt many medical practitioners keep their books on a proper double entry system and prepare an income account periodically. These should find no difficulty in preparing their returns; it is to those who have not done so and who are not well versed in the necessary accountancy knowledge required for the preparation of the return, that the following remarks are specially directed.

It is not possible in the space available to refer to each item of "income" and "deductions" in the order in which they appear on the returns, as the forms supplied by the taxation departments in the various States differ somewhat in regard to the details thereon and the order in which the items appear.

I will therefore refer briefly to the several classes of income and deductions which should be included in the return, leaving to each individual taxpayer to show his income and deductions alongside the appropriate items on the form of return supplied by the taxation department in the particular State in which the income is earned.

Forms to be Used.

A medical practitioner, practising his profession on his own behalf in Australia, will prepare a return of his income on a form supplied by the taxation department of his State and described in the various States as follows: New South Wales, Queensland and Victoria, "Form A"; South Australia, "Form 2"; Western Australia, "Form H"; and Tasmania, "Form 5."

When two or more persons are practising in partnership, a return will be prepared for the whole of the partnership transactions on one of the forms referred to above.

The net income shown on the partnership return is not taxable by the department against the partnership; each individual partner is taxable on his share. Details as to the names, place of residence, proportionate share of income to which each partner is entitled, amount of each share and in certain States the relationship of each partner one to another must be given in a separate statement contained on the return.

Each individual partner must then prepare a separate return described in the various States as follows: New South Wales and Victoria, "Form A"; Queensland, "Form B"; South Australia, "Form 1"; Western Australia, "Form HA"; and Tasmania, "Form 5." Each partner must show the amount of his share as set out in the appropriate statement on the partnership return, together with any income received from other sources, except interest on State Government bonds, stock or debentures issued prior to January 1, 1924, which is not taxable.

A medical practitioner not practising his profession on his own behalf, but in receipt of a salary, will use similar forms to those described in the immediately preceding paragraph.

If a taxpayer derives income, other than dividends, from more than one State, he must also furnish a return of his income on a special "Federal Income Tax" form to the Deputy Federal Commissioner of Taxation in Melbourne. In such case the combined Federal and State return referred to above should be forwarded to the State Commissioner of Taxation, endorsed "For State purposes only."

Income.

Professional Income. Professional income includes the amount of gross income from practice, by way of fees, lodge payments and medicines supplied during the year.

If a proper record has been kept of all fees earned during the year this figure will be readily arrived at. If no record has been kept, but all fees *et cetera* have been banked, then the bank pass book will show the amount of gross income for the year.

If all fees *et cetera* have not been banked, then to those actually banked must be added the amount retained and used for expenses, either private or professional.

When fees *et cetera* are used for professional expenses without being passed through the bank account, I would suggest that in future a book be kept for recording such expenses, as it is quite probable that the department may not allow the expenses as a deduction from income unless details are supplied.

Dispensary. If a medical practitioner conducts a dispensary and if the dispensary is run entirely apart from his medical practice, it is, of course, essential that proper records be kept of purchases and sales of medicines *et cetera*, wages and other expenses, if any, in connexion therewith, and that the stock of medicines *et cetera* on hand be listed and priced at June 30 in each year.

These items of revenue and expenditure in connexion with the dispensary would not be shown on the return as income *et cetera* from the practice, but in the appropriate portion of the return provided for the transactions of a business.

Rent Revenue. When a medical practitioner pays rent for the premises in which he is carrying on his practice and when any portion of such rented premises is used as a residence, an amount equal to two-thirds of the total rent paid should be treated as income. As the whole amount paid as rent will be claimed as a deduction, the net allowance for rent in connexion with the professional practice is thus equivalent to one-third of the total rent paid.

If it is considered that one-third of the total rent paid is too small a proportion to be allowed in the particular circumstances, the amount which the taxpayer considers fair and reasonable should be stated together with the grounds on which he arrives at such amount.

Bad Debts Recovered. Only such amounts as have been actually written off as bad and claimed as a deduction from income in previous years and which have been recovered during the year covered by the return, should be included under the heading of bad debts recovered.

Care should be taken by medical practitioners who do not keep a complete set of books, to see that such items are not also included in the gross income referred to above, otherwise double taxation will result.

Deductions.

The following items of expenditure are allowed as deductions from income for both Federal and State purposes, except when specifically stated to the contrary.

Salaries and Wages. The amount of salaries and wages actually paid to any person in the employ of the taxpayer who is engaged in work necessary for the production of his income, is allowed as a deduction. Such persons include an assistant doctor (on a salary), a nurse, a *locum tenens*, a charwoman for cleaning consulting rooms (if rooms are cleaned by a general servant, portion of her wages can be claimed as a deduction under this head), a chauffeur, a groom.

Food for Employees. A medical practitioner who employs any person exclusively for the purposes of his practice and in addition to the wages or salary of such person paid in money, supplies him or her with food, he may claim as a deduction the amount actually expended thereon. If in these circumstances the taxpayer is unable to state the actual amount expended as aforesaid, then such sum is deducted as in the opinion of the Commissioner is just and reasonable.

Rent. The amount of rent paid for consulting rooms, or if a medical practitioner is practising at his place of residence rented by him, then the total amount of rent paid for such residence is deductible. (Note reference to this item under heading "Rent Revenue.")

Rates and Taxes. If a medical practitioner is renting or leasing property and is using the property as a res-

dence and consulting rooms combined and if the lease provides for the payment by the tenant or lessee of municipal and/or water rates, one-third only of such rates (not including excess meter rates) actually paid during the year can be claimed as a deduction for New South Wales, South Australian and Tasmanian State purposes, while the full amount actually paid can be claimed for Queensland, Victorian and Western Australian State and also for Federal purposes.

If the leased premises are wholly used as consulting rooms the total amount actually paid can be claimed as a deduction for all States as well as Federal purposes.

Gas, Electricity and Telephone. A proportion of the cost of gas or electricity or other means of lighting as would refer to that used in connexion with the medical practice may be claimed as a deduction together with the amount paid for telephone during the year.

Insurance. Any fire or burglary insurance premiums paid for the insurance of any property used in conducting the practice, such as office, consulting and waiting room furniture, surgical instruments and plant, motor car, horses or vehicles, together with workmen's compensation premiums, may be deducted.

Interest. The actual amount of interest paid on money borrowed for the purpose of acquiring or carrying on a practice may be claimed as a deduction under the heading of interest.

Depreciation. A deduction may be claimed under the heading of depreciation for the wear and tear of property (other than land and buildings) used for the purpose of carrying on the practice. The rate of depreciation allowed may vary slightly as regards the several State departments, but the following rates are those allowed for Federal purposes:

- | | |
|---|-----|
| (a) Doctors' instruments: No depreciation, but the cost of replacements (not including alterations, additions or improvements) is allowed under the heading of "repairs." | |
| (b) Electrocardiograph | 5% |
| (c) Furniture and fittings (consulting rooms) .. | 2½% |
| (d) Furniture and fittings (hospitals) | 7½% |
| (e) Carpets | 10% |
| (f) Ophthalmic surgeon's machinery | 10% |
| (g) Motor car | 10% |
| (h) Motor cycle or bicycle | 10% |
| (i) Röntgen ray machines | 7½% |
| (j) Typewriter | 10% |
| (k) X ray and high frequency current plant .. | 7½% |
| (l) Buggies and sulkies | 10% |
| (m) Horse | 10% |
| (n) Horse rugs | 20% |
| (o) Harness | 10% |
| (p) Buildings (for Queensland State purposes only), 2½% wooden, 1% brick <i>et cetera</i> . | |

Note that depreciation can be claimed only as a deduction at the above rates, calculated on the diminishing value of the assets as at June 30 of the previous year, that is, the cost price of the asset, less the above percentages of depreciation deducted for each year in use in the business.

No depreciation is allowed on any of the above purchased during the year on which the income return is based.

Repairs. All repairs and replacements (which would not come under the heading of alterations, additions or improvements) effected by a medical practitioner at his own cost to any assets employed in carrying on the practice, including covenanted repairs to that part of rented premises used as consulting rooms, are allowed as deductions.

Bad Debts. If all fees earned have been previously returned as income, irrespective of whether they have been collected or not, and any of such fees still remain uncollected and are irrecoverable and have been actually written off as bad debts, such amount will be allowed as a deduction from the current year's income.

If in the past and also in the present return only the actual fees received have been included as income, then, of course, no deduction can be claimed for bad debts.

Exchange and Discount. The heading "exchange and discount" will hardly be affected by any transactions of a medical practitioner except in so far as any exchange paid on country cheques is concerned.

Travelling Expenses. Only travelling expenses incurred in the production of income will be allowed as a deduction, for example: (i) motor car expenses, cost of running, benzine, oil, tires, garaging *et cetera* (repairs to be shown under separate heading referred to above); (ii) buggy and sulky expenses, horse feed, shoeing *et cetera* (harness and vehicle repairs to be shown under heading "Repairs"); (iii) railway, tram, boat, taxi-cab and cab fares or the cost of hiring a vehicle for conveyance to or returning from a professional visit.

As regards Western Australian State income tax, travelling expenses from a taxpayer's residence to his place of business (not exceeding £15) are allowed as a deduction.

Stationery, Stamps *et cetera*. The cost of all printing, stationery, stamps and telegrams incurred in connexion with the practice will be allowed as a deduction.

Other Business Expenses. Any other expenses, not elsewhere included, necessarily incurred in the production of income should be shown under this heading, for example: (a) cost of bandages (when no dispensary is kept), (b) cost of chemicals (when no dispensary is kept), (c) accountancy fees, (d) bank charges, cheque books, *et cetera*.

State Income Tax. The amount of State income tax paid during the period covered by the return is allowable as a deduction for Federal purposes only.

Protection of Income. Expenses incurred in the protection of income when such income cannot be insured, are allowable as a deduction for State purposes in Western Australia, but not in any other State nor for Federal purposes.

Federal Income Tax. The amount actually paid by a taxpayer during the year for Federal income tax is allowed in Western Australia only for State purposes; it is not allowed for Federal purposes.

Additional Transactions.

I have enumerated above the various items of income most generally received and expenditure incurred by a medical practitioner in carrying on his professional practice. Several other items of income and expenditure *et cetera* may be mentioned which may not necessarily affect a medical practitioner's return in so far as his professional transactions are concerned; it should be specially noted, however, that the following are liable to taxation or may be claimed as deductions.

Concessional Deductions.

Calls on Shares. The total amount of calls paid during the year covered by the return to companies and syndicates mining in Australia for gold, silver, base metals, rare minerals or oil, or carrying on afforestation is allowed as a deduction. This is for Federal purposes only. It should be noted, however, that in Queensland calls paid to companies and syndicates carrying on mining operations in Queensland or afforestation as the principal business, and that in Victoria calls paid to companies and syndicates carrying on mining operations in Victoria are allowed as deductions for Queensland and Victorian State purposes respectively.

Application and Allotment of Moneys.

Application and allotment moneys paid in respect of shares in companies are not calls and do not come under this heading.

Application and allotment moneys paid to companies and syndicates carrying on mining operations in Queensland or afforestation as the principal business are allowed as deductions for Queensland State purposes only.

Gifts. Gifts of £1 and upwards to public charitable institutions and public universities (or colleges affiliated thereto) in Australia and to public funds for a Commonwealth or State war memorial are allowed as deductions for Federal purposes only.

The New South Wales, Victorian, Queensland and Western Australian State Taxation Departments allow as

a deduction gifts (exceeding a sum specified on the return) to public charitable institutions within the particular State.

Life Assurance Premiums. Premiums paid by a medical practitioner in Australia on the insurance of his own life or that of his wife or children are allowable deductions. The maximum amount allowed for Federal purposes is £50. New South Wales, Queensland and Western Australian State departments also allow this deduction, while Victoria allows the deduction only in respect of a premium on the taxpayer's own life. South Australia and Tasmania do not make any allowance for this item.

Deduction for Children. The sum of £50 is allowed for Federal purposes for each child under the age of sixteen years at the commencement of the year covered by the return and wholly maintained by the taxpayer; a proportionate amount is allowed in respect of a child who reaches the age of sixteen years or who is born during the period covered by the return, provided that no deduction is allowable in respect of any child who earned income during the year.

A deduction under this head is also allowed by each of the State departments. The amount allowed in respect of each child varies in the different States, and as regards Victoria and Queensland is subject to the taxpayer's net income not exceeding a specified amount, while in Queensland the amount allowable is calculated on a sliding scale.

The actual amount allowed by each State department and the qualifications (if any) regarding such allowance are set out in detail on the returns issued in the various States.

Deduction for Wife. No deduction is allowed in respect of a taxpayer's wife for Federal purposes. New South Wales, Queensland, Victorian and South Australian State departments allow a deduction of varying amounts in this connexion, but the deduction is subject to the taxpayer and also his wife not being in receipt of a certain specified income.

As in the case of the deduction for children, all particulars of allowances *et cetera* are clearly set out in schedules embodied in the returns issued in each State. No deduction is allowed in Western Australia or Tasmania.

Maintenance of Dependents. The amount expended by an unmarried taxpayer towards the maintenance of his dependants is allowed by New South Wales, Queensland, South Australian and Western Australian State departments. The amount of deduction allowed and the information required vary considerably in the different States, but the statement embodied in the return is very explicit in each instance and requires no further explanation.

No allowance in this respect is made for Federal purposes or for Victorian and Tasmanian State purposes.

Medical and Funeral Expenses. Amounts paid to any legally qualified medical practitioner, public or private hospital, nurse or chemist on account of illness of taxpayer, his wife, or children under twenty-one years of age, and on account of funeral expenses paid (not exceeding £20) in respect of the death of the taxpayer's wife or any of his children under twenty-one years of age are allowed as deductions. This deduction is allowed for Federal purposes only when the taxable income does not exceed £900.

The New South Wales, Queensland, Victorian and Western Australian State Departments also allow this deduction, but it is subject to the taxable income not exceeding a specified amount as shown in detail on the returns supplied in the various States.

As regards the New South Wales State Department, medical and hospital expenses and payments to a nurse or chemist on account of illness of a taxpayer's children over the age of twenty-one years and on account of dependants of the taxpayer are also allowed as a deduction.

Dental Expenses. Amounts paid to any legally qualified dentist are allowed by the New South Wales Taxation Department only.

Donations. Donations for research into the causes, prevention or cure of disease in human beings, animals or plants, to any public authority engaged in such research, are allowed as a deduction for Federal purposes only.

Education of Children. Expenses actually incurred by a taxpayer (not exceeding £50 for each child) in educating his children under the age of eighteen years are allowed as a deduction by the New South Wales State Taxation Department when suitable educational facilities are not provided by the State within reasonable travelling distance of the place of residence of the taxpayer. This deduction is not allowable if the taxpayer's taxable income exceeds £800.

Income from Property.

Rents. The gross amount of rent received from properties, including rent from subletting, must be shown alongside this heading. A detailed list giving the source of such revenue must accompany the return.

Dividends. The gross amount of all dividends from companies in all States, including shares distributed as bonus shares, must be shown under the heading of dividends. As regards Queensland and Western Australian returns, a similar amount will also be shown under the heading of dividends amongst the list of deductions, for State purposes only.

Any rebates allowable on account of income having been earned by the company outside the State or Australia or having been taxed in the hands of the company will be calculated and deducted by the department.

Interest Received. Particulars of all interest received from mortgages, deposits of all kinds, Savings Bank deposits, also from bonds, stocks or debentures issued by companies or public bodies and by a State Government or by any State authority after December 31, 1923, and from all taxable Commonwealth loans must be shown in detail on statement embodied in the return. These are all taxable for Federal purposes. The items included in the above which are taxable for State purposes, are enumerated on the returns of each individual State.

Own Residence. When a taxpayer resident in Victoria owns property (including property being purchased on terms) and he uses such property for the purpose of residence or enjoyment, he shall return as income 4% of the capital value of such land and improvements thereon. This is for Victorian State purposes only.

Deductions from Property Income.

All expenses actually incurred in gaining or producing the assessable income from property are allowed as a deduction therefrom, for example:

- (i) Water and sewerage rates, State and Federal land tax and fire and burglary insurance premiums paid on income-producing property.
- (ii) Repairs (not including alterations, additions or improvements) to properties from which rent is received.
- (iii) Commission paid for collection of income from property.
- (iv) Interest paid on borrowed money used to produce income from property from which rent is received.
- (v) Interest paid on a mortgage of land and residence owned or in course of purchase and occupied by a taxpayer and in respect of which 4% of capital value is returned as income (allowable for Victorian State purposes only).
- (vi) Rent paid for property from which rent is received.

The above-mentioned deductions (in some cases similar to those previously mentioned in this article) apply only to that portion of such expenditure which refers to income received from property, and will be claimed under this section of the return by taxpayers having income-producing investments outside their professional practice.

Expenses Not Allowed as Deductions.

The following items of expenditure are not in any circumstances allowed as deductions from income for either State or Federal purposes, except where specially mentioned:

- (a) Additions or alterations to trade or other income-earning premises.
- (b) Additions to plant and machinery (except in mining businesses under certain conditions).
- (c) Any domestic expenditure or the cost of living of members of the taxpayer's family not exclusively engaged in the business or who are engaged in domestic duties.

- (d) Cost of sewerage connexions.
- (e) Cost of travelling between taxpayer's private residence and place of business (except by Western Australian State Department).
- (f) Depreciation of any kind that may be made good by repairs.
- (g) Depreciation of goodwill.
- (h) Doubtful debts.
- (i) Expenditure incurred to protect income (except by Western Australian State Department).
- (j) Federal income tax (except by Western Australian State Department).
- (k) Insurance (fire or burglary) on household furniture or personal effects.
- (l) Interest not actually incurred in the year covered by the return.
- (m) Interest paid on money which is not used to produce income (except for Victorian State purposes, on taxpayer's private residence).
- (n) Losses by fire, accident, robbery or embezzlement.
- (o) Losses not connected with or arising out of the taxpayer's trade or business.
- (p) Purchase money paid, except for trading stock.
- (q) Payments from husband to wife or from wife to husband, unless the Commissioner is satisfied that the payments have been made *bona fide* in the course of business and for services rendered.
- (r) Premiums on insurances effected outside Australia.
- (s) Rent of private residence.
- (t) Repayment of moneys borrowed, including amounts of principal included in annual payments on loans.
- (u) Wages to persons not employed in the trade or business.
- (v) Repairs, insurance, interest paid and other expenses incurred in connexion with taxpayer's private residence (excepting rates and taxes which are allowed for Federal purposes and interest for Victorian State purposes).

Preparation of Return.

The Federal and State (combined) income tax forms supplied by the taxation departments in the various States, although differing somewhat as regards wording and the order of items thereon, are all divided into four parts, headed A, B, C and D respectively, with the exception of the New South Wales return, which is divided under two headings only, namely, "Income Derived from Personal Exertion" and "Income Derived from Property."

As regards income earned in either New South Wales, Victoria or Tasmania, it will be necessary for each taxpayer in these States to prepare a statement of his professional income and allowable deductions therefrom (not including concessional deductions). The net result of these items of income and deductions will represent the net income from the practice of the profession, and this amount will be shown in the appropriately worded line of Part A of the return as regards Victoria and Tasmania and under the heading of "Income Derived from Personal Exertion" on the New South Wales forms.

The statement of professional income and deductions referred to above must, of course, be attached to and lodged with the return.

Concessional deductions will in each instance be shown on a separate portion of the return in space specially provided.

As regards Queensland, South Australian and Western Australian returns, I would suggest the preparation of a similar statement to that required by the other States before the return is filled in. It is not necessary for this statement to accompany the return, but each amount of professional income and deductions (including concessional deductions) should be shown alongside their respective items in Part C of the returns in each of these three States.

The above directions apply equally to the transactions of a professional practice carried on by two or more medical practitioners in partnership. In such circumstances the individual partners will also furnish a separate return and show their respective shares of the net income as set out on the partnership return alongside the item "Share in partnership of" in Part A of the returns

of all States except New South Wales, where it is described as "Share of profits derived from the partnership of . . ." under the heading of "Income Derived from Personal Exertion."

Medical practitioners not in practice on their own behalf but working for a salary will show the amount of such salary received during the year ended June 30, 1931 (including any allowances, bonuses or overtime) alongside the appropriately worded item in the portion of the return marked Part A (in New South Wales, "Income from Personal Exertion").

When the return has been completed to the extent referred to above, all transactions in connexion with the professional practice should appear thereon.

It will now be necessary in every instance where a medical practitioner is in receipt of any income from investments outside his medical practice to prepare a list of such income and allowable deductions therefrom and to show the total of each item in its respective place in Part B of the returns in all States except New South Wales, where the headings appear as "Income Derived from Property" and "Deductions in Connection with Income Derived from Property."

It will be noted that the form of return issued in each State is provided with two columns alongside each set of items; these are for Federal and State purposes respectively, according to the headings.

It should also be noted that where items are not allowed as deductions in certain States, but are allowed for "Federal purposes only" or *vice versa*, it will be necessary to show the amount of such item on the return, but extended into the appropriate column only (Federal or State, as the case may be).

In all States it is necessary for the taxpayer to show each item of income and deductions in both columns when the income and deductions are for Federal and State purposes, or in the appropriate column when the item of income is taxable or the deduction is allowable for Federal or State purposes only.

Both columns should be added up and the amount of net income taxable for Federal and State purposes shown.

Finally, the return and all accompanying schedules, if any, should be signed personally by the taxpayer, when possible, and lodged with the taxation department in the respective States on or before a date advertised in each State and also shown on the form.

In most States the dates for lodgment are usually July 31 for persons in receipt of salary only and August 31 in the case of medical practitioners practising their profession either in partnership or on their own account.

Verification of Deductions.

All deductions in respect of donations, gifts to charitable institutions *et cetera*, medical, dental (New South Wales) and hospital expenses should be supported by receipts, otherwise the deductions may be disallowed.

Statements on Return.

The returns in each State contain several statements which must be filled in by the taxpayer if any deductions are claimed in respect to the headings printed thereon, namely:

- Salaries, wages and commission paid and allowances to employees.
- Statement by trustee or partnership.
- Deductions claimed in respect of children.
- Dividends from companies.
- Particulars of taxable interest received.
- Deductions claimed for medical and funeral expenses (Queensland, Victoria and South Australia only).
- Rates and taxes (Western Australia only).
- Deduction for wife (New South Wales, Victoria and South Australia only).
- Depreciation (South Australia only).
- Contributions to dependants (New South Wales, Queensland, South Australia and Western Australia only).
- Transfers of property by married persons (New South Wales only).

The remaining statements on the various returns are not likely to be affected by the ordinary transactions of a medical practitioner, but should they apply in any particular instance the necessary information must be supplied.

Detailed Lists Required.

Wherever deductions are claimed on account of any items in respect of which the form calls for a detailed list, such list must be attached to the return, otherwise the deductions may be disallowed.

Compliance with the above direction may also save the taxpayer much correspondence and irritation.

Except in certain States where a special form is embodied in the return, separate detailed lists of the following deductions are necessary:

- Gifts, charitable contributions *et cetera*.
- Donations for research in connexion with causes, prevention and cure of diseases.
- Calls in mining companies.
- Fire and burglary insurance premiums.
- Medical expenses *et cetera* (form of statement contained on the returns in South Australia, Victoria and Queensland).
- Rates and taxes (form of statement on Western Australian return).
- Repairs.
- Depreciation (items, values and rates of depreciation and manner in which same is calculated).
- Bad debts (written off), giving date incurred and amount.
- Contributions to employees' benefit or provident fund.
- Other business expenses.
- Travelling expenses.
- Application and allotment moneys (Queensland).

Copy of Return.

A copy of the return, together with books, accounts, memoranda and all data from which the return has been compiled, should be kept for future reference.

General.

In conclusion, it might be pointed out that the above explanations and directions have been confined to the general transactions of a medical practitioner in the ordinary course of his practice; they have been supplemented by a few remarks regarding concessional deductions together with certain items of income from property, which may possibly affect the return.

Many fine legal points that may arise in such rare instances as when extraneous transactions have occurred, have not been dealt with (in such cases special inquiry should be made at the department of taxation in the respective States). Apart from these, I believe that, should the above directions be carefully followed, the full amount of deductions as allowed by the Federal and various State income tax acts will be obtained.

I have endeavoured to be as concise and explicit as possible and trust that the above, when read in conjunction with the return itself, will prove of some assistance to the medical practitioners of Australia in preparing their income returns for the year ended June 30, 1931.

ROBT. J. STIFFE, A.C.A. (Aust.).

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Mooroopna on October 11, 1930.

The Stomach from a Radiological Aspect.

DR. J. W. FLORANCE read a paper entitled: "The Stomach from a Radiological Aspect." He described the normal and abnormal conditions of the organ as to its form, its tone,

motility and peristalsis. He discussed the position and mobility both in the prone and upright patient. He also referred to spasm and noted its significance, whether it caused the hour glass stomach or whether it was diffuse and caused distortion. He referred to its frequency at the pylorus involving the question of its extrinsic or intrinsic origin. Films were shown, notably one of ulcer followed by excision. Cases of cancer of the stomach in which absence of filling was marked, were shown on films.

DR. COLIN MACDONALD congratulated Dr. Florance on his paper. He stressed the importance of adequately accommodating the eyes before commencing screening. He felt that a standardized carbohydrate motor meal was of value; in his experience a gastric residue after six hours was strongly suggestive of an organic lesion of the stomach, duodenum or gall bladder, and he regarded this as the most important of the "indirect" signs of gastro-duodenal pathological change. He disagreed with some radiologists of international repute who held that a history with the biochemical findings was unnecessary. Time, trouble and expense to the patient were often saved if these data were available to the radiologist before the X ray examination.

Clinicians should be aware of the fact that the radiological examination of adipose patients presented many more difficulties than in people of slender build. In the former variety more time and X ray material were expended and diagnosis was rendered more difficult. It was in these patients that the use of the Bucky fluoroscopic grid had proved of extreme value.

He felt that radioscopy examination should never be rushed through and completed in a few minutes. To exclude small lesions from every segment of the stomach, careful screening in all positions, from the erect to the Trendelenburg, was necessary.

The right lateral prone position, described by Carman some years ago, had proved of great value in outlining the duodeno-pyloric area and had robbed this area of a lot of its previous uncertainties. Both Dr. H. M. Hewlett and he were very rarely satisfied with radioscopy alone, and nearly always took numerous films. They paid particular attention to obtaining satisfactory serial films of the pyloric region.

The work of Berg and Forsell on the outlining of the mucosal rugae, which had been introduced to Melbourne by his colleague, Dr. O'Sullivan, promised to be of value in the earlier detection of gastric lesions.

The question of gastric diverticulation had to be considered when a niche projecting from the lesser curvature was seen. While these were most frequent at the cardiac end, Dr. Hewlett recently had a case in which the diverticulum lay silhouetted against the lesser curvature border in the middle third of the stomach, in the usual site for a chronic penetrating ulcer.

The radiologist was sometimes asked to give an opinion on the resectability of a gastric carcinoma. He felt that this was often impossible. While radiologically the position and approximate extent of the growth could be demonstrated, as well as its fixation to other organs, operative findings generally showed that the neoplastic infiltration extended much further than the screen or films indicated, and little information could be obtained as to the presence of secondary invasion.

He had rarely seen chronic penetrating gastric ulcers in the immediately prepyloric position, nor had he seen them at the cardiac end above the level of the oesophageal opening.

A patient whose stomach he had recently examined by X rays with normal findings, wrote from a Swiss sanatorium protesting that he had overlooked a large ulcer which the patient claimed had been subsequently demonstrated by a Swiss radiologist on the greater curvature at the cardiac end under the left diaphragm and above the level of the oesophageal opening. He asked the surgeons present if they had ever seen an ulcer in this situation.

The interpretation of findings in the anastomosed stomach often presented difficulties. In his experience a common finding in patients who were examined by X rays for a return of their dyspeptic symptoms some months after gastro-enterostomy, was a six-hourly residue in the pyloric end of the stomach, between the stoma and the

pylorus, even though the stoma was functioning well and the meal was also passing *via* the pylorus. He wondered whether this was solely a mechanical phenomenon, due to the stoma being placed too high (though he appreciated the anatomical difficulties of placing the stoma along the greater curvature border) or was it due to pyloric spasm occurring at the end of digestion, the result of a recurrence of the duodenal ulcer for which the anastomosis was originally performed?

He had seen recurrence of a chronic penetrating ulcer niche at or near the site of excision of a previous ulcer, indicating the strong ulcer diathesis exhibited by some patients.

The question of malignant degeneration around a chronic non-malignant ulcer was bound up radiologically with that of "ulcer-tumour"; when a chronic ulcer niche was observed to be surrounded by a persistent irregular filling defect, the problem confronted the radiologist as to whether this was originally a simple ulcer on which malignancy had supervened, or ulceration in a primary malignant condition, or an "ulcer-tumour" due to inflammatory thickening around a simple ulcer. It was sometimes practically impossible to make a diagnosis on the X ray evidence alone. In the few such cases he had seen, the length of history appeared to eliminate malignant disease.

The association of gastric or duodenal ulcers ought not to be forgotten by the radiologist, but he had never seen a dual lesion in as high a percentage as had been claimed in other quarters. Finally, he rarely relied on "indirect" signs in gastro-intestinal radiology, and the majority of surgeons nowadays required "direct" demonstration of a lesion before resorting to surgical operation.

In reply, Dr. Florance said that his paper had been worth reading, if only to induce Dr. Macdonald to join in the discussion.

Swift's Disease.

Dr. C. H. DICKSON read a paper entitled: "Swift's Disease" (see page 14).

Dr. H. BOYD GRAHAM congratulated Dr. Dickson on his paper and said that there was very little to add. Referring to the view that erythroedema was probably a deficiency disease, Dr. Graham was of opinion that this would be proved to be the case. The age incidence was against its being an infection only. There were at least three instances of more than one child in the same family having the disease at different times. It was not due to the deficiency of any known vitamin.

McCollum, in his book, "The Newer Knowledge of Nutrition," quoted an experiment in which a disease similar to pink disease was produced in rats fed on a diet normal to rats, except that dried white of egg was given instead of raw white of egg. The disease could be prevented by giving either raw white, egg yolk or raw liver.

The vitamin was probably closely related to vitamin B and was probably sensitive to heat. To clear up this point clinically it was important to apply the therapeutic test in early cases by making no change in the diet except to give food containing one or all of the elements described above.

In cases which occurred in children who were on the breast, it was probably advisable to wean the child at once, as the mother was usually poor and unable to feed herself properly.

Dr. H. DOUGLAS STEPHENS expressed appreciation of Dr. Dickson's very comprehensive paper. He (Dr. Stephens) had been associated with the late Dr. Snowball when the illness had been recognized as a clinical entity and given the name of the "raw beef disease."

It presented some of the characteristics of an infectious disease, as eighteen or more cases might be seen by one observer in one season and then there might be none for nearly a year. Dr. Stephens had never seen more than one case in the same family at the same time. Dr. Stephens referred to the experimental work which he had recently seen in England; Dr. Boas said that yeast would ward off the disease in rats, but that "Marmite," which was prepared from yeast, had no protective effect. With the Melanbys at Sheffield he had seen the results of experi-

ments in cases of puerperal sepsis with what they called the anti-infective vitamin, "Radiostolum."

He had himself tried "Radiostolum" in pink disease in four cases. In three cases there was no result, but the fourth child rapidly recovered. It had been ill for three months and might have been convalescent in any case.

The age incidence ranged between four or six months and four years. One of his patients, a boy aged two and a half years, had been very ill with all the septic manifestations. He (Dr. Stephens) had tried all the usual remedies without much success, including unboiled milk in large quantities given with a stomach tube, and cod liver oil emulsion. After an injection of antistreptococcal serum this child's illness had cleared up in a fortnight. In other similar cases serum had had no result.

Dr. Stephens had gained the impression that the average length of the illness had lessened and the prognosis improved since the introduction of the modern methods of vitamin administration.

In some patients whom he had seen in England, real anaesthesia had occurred. One child had bitten off the ends of all its fingers and toes. Dr. Stephens regarded the disease as not due to an infection and not due to a toxin acting on the endocrine or any other system, but due to the deficiency of a specific vitamin.

Eclampsia.

Dr. F. GRÜTZNER read the clinical notes of three cases of eclampsia (see page 17).

Dr. IVAN MAXWELL congratulated Dr. Grützner on his interesting paper. Referring to the biochemistry of eclampsia, Dr. Maxwell said the fact that there was no albuminuria in the second case suggested that there was no renal inefficiency. Routine urea concentration and blood urea estimations were valuable in treating the condition, the latter being the more valuable, but more difficult to carry out except in a well equipped laboratory. The Hensch-Aldrich test for the estimation of blood urea required the withdrawal of eight cubic centimetres of blood from a vein, was easily performed and would throw light on such cases, especially in the country.

Cholecystography.

Dr. A. L. BENNETT read a paper entitled: "The Value of Cholecystography in Diagnosis" (see page 12).

Dr. W. E. DONALDSON congratulated Dr. Bennett both on her paper and on the work it described. He agreed with her in regard to the difficulty experienced in examining very stout people with X rays. Even if the patient did eventually vomit, a good shadow usually could be obtained so long as the dye was retained for from ten to fifteen minutes.

Figures from Seattle showed that 21% of disease conditions occurring in the upper part of the abdomen were due to gall bladder disease. A well visualized gall bladder shadow did not necessarily exclude a pathological condition of the gall bladder and normal X ray findings in the presence of positive clinical evidence should not usually be taken to exclude gall bladder disease.

Dr. VICTOR HURLEY praised the paper as representing a record of work done. The results of cholecystography were becoming more and more reliable. Dr. Clendinnen's radiological department at the Melbourne Hospital had recently recorded series of results showing a diagnostic accuracy of more than 90%.

As to the verification of suspected gall bladder disease, this was not always easy, even at operation. A gall bladder might seem almost normal on being handled, yet after removal might prove to be a typical "strawberry" type and might even contain small pigment stones. Gall bladder disease might simulate other diseases. To illustrate this point, Mr. Hurley quoted several cases, notably that of a business man who suffered for some years with spinal and lumbar pains which were cured by removal of his gall bladder. Similarly recurring attacks of colitis might be dependent on chronic cholecystitis.

Dr. Hurley described Ewatts Graham's method of using the dye test for hepatic efficiency and said that this work was now being done in Melbourne. Patients suffering

from stone in the common duct of long standing presented a much greater operative risk than was obvious to ordinary examination, owing to their reduced hepatic efficiency.

DR. COLIN MACDONALD expressed pleasure that Dr. Bennett had so sanely and temperately stated the case for cholecystography which was now generally regarded as a test of very great value. He felt that cholecystography probably had, up to date, served its best function in greatly increasing the percentage of gall stones demonstrated. He was not able to confirm entirely the hopes originally entertained in certain eminent quarters that this investigation was a means of diagnosing early pathological change in the gall bladder. He could not subscribe to high claims of percentage accuracy (in the region of 95%) advanced by some radiologists abroad; he appreciated the fact that some of these men were using the intravenous method of administration, thereby eliminating certain sources of error from the intraoral method, which was uniformly used in Melbourne.

He believed that the varying claims for the percentage accuracy of the test were bound up with the varying surgical and pathological criteria of gall bladder disease.

There were certain difficulties which had confronted him in the interpretation of cholecystograms: (i) Whether the dye had been adequately absorbed from the alimentary tract. With the adequate dose of the improved dye it was generally accepted that this was nowadays rarely a source of error. (ii) The fact that 50% of patients with mild, non-calculous cholecystitis gave normal cholecystograms. (iii) Owing to the scattered and secondary rays produced in adipose patients, the gall bladder shadow cast was never of the brilliant white density that obtained in thin patients, and so the question as to whether concentration was normal or subnormal was always a difficult one. (iv) It was sometimes impossible, especially in hypersthenic patients, to project the colonic or duodenal contents away from the fundus of gall bladder shadow. This overlapping produced a "mottling" often difficult to differentiate from gall stones. He found the erect posture the most satisfactory in eliminating this difficulty. This erect posture, too, would sometimes demonstrate gall bladder sand at the fundus, which would not be demonstrated if the films were taken in recumbency. (v) There were several conditions unassociated with pathological gall bladders which caused non-filling. It had been found that extensive hepatic disease had to be present to prevent excretion of the dye. (vi) Sometimes in hypersthenic patients the gall bladder shadow might lie behind the vertebral column when films were made in the ordinary postero-anterior position. To exclude this error, films in various positions of rotation had to be exposed.

In conclusion, Dr. Macdonald joined with Dr. Florence in stressing the great importance of correct X ray and photographic technique in cholecystography.

Carcinoma of Oesophagus.

DR. R. O. MILLS showed a man, seventy-one years of age, whose history was that of oesophageal obstruction of fairly sudden onset four months previously. Partial relief had been obtained for a time by reducing superadded spasm by the use of atropine. Clinical examination had failed to show any extrinsic cause for the obstruction, but screening with a barium meal demonstrated clearly the irregular carcinomatous obstruction at and below the level of the bifurcation of the trachea. Several radiographs taken at different periods were shown. Dr. Mills said that gastrostomy had been performed four weeks previously under ethylene anaesthesia, with the result that the general condition of the patient was being satisfactorily maintained.

Mitral Stenosis with Recurrent Hæmoptysis.

DR. J. A. KENNEDY showed a male patient, aged twenty-nine years, whose illness commenced eight years previously. He was crushed playing football, played throughout the match and had a small hæmoptysis that night. Following this he suffered from small hæmoptysis after indulging in any sport. Severe hæmoptysis occurred one year ago. Since that time dyspnoea had occurred on slightest exertion. No tubercle bacilli were found in

the sputum. The Wassermann and Casoni tests gave no reaction. Examination of heart revealed mitral stenosis.¹

DR. S. O. COWEN said it was a question whether repeated hæmoptyses over such a long period of time could be due to mitral stenosis alone. The skiagram of the lung indicated some peribronchial fibrosis which was consistent with mitral stenosis, but the possibility had to be considered of a low grade tuberculous lesion. The temperature should be observed and the sputum examined for tubercle bacilli many times up to twenty.

Chronic Ulcer of Leg: Sympathectomy.

DR. W. L. ARMSTRONG showed a woman, aged thirty-nine years, who since the age of eleven years had had some swelling of the legs. During her thirteenth year she had two operations for removal of the tonsils, followed by pneumonia. At the age of sixteen years, just before menstruation commenced, her legs became swollen. The swelling was thought to be endocrine in origin and the patient was treated with thyreoid. Menstruation was comparatively regular in the early stages, but for some years the patient had periods of amenorrhœa of about three months' duration. The patient's mother suffered from rheumatoid arthritis. Her sister's child died at the age of ten years from Addison's disease. Pelvic examination and X ray examination of the spine and pelvis revealed no abnormality. The Wassermann test gave no reaction. About three and a quarter years ago an ulcer formed and has been present intermittently since. The patient spent September and October in hospital. In January, 1929, she suffered from myositis of the back. After resting for about nine weeks in hospital the ulcer healed, but one week after the patient returned home it broke down and persisted in spite of treatment and, increasing rapidly in size, formed an excavated ulcer of the diameter of half a crown, with black sloughing base, becoming very painful and obviously involving the periosteum. On August 19, 1930, sympathectomy was performed by the combined technique of Leriche and Handley on the femoral artery in Hunter's canal. An immediate and appreciable alteration in the warmth of the affected foot and leg took place on the table and was continued during the patient's stay in hospital. The patient stated that the leg was comfortable and the improvement in colour from a cold, marble-like appearance to that of healthy pink was marked with definite diminution in size. Rapid repair took place and was complete in three and a half weeks. The limb now remains diminished in size, painless and warm.

DR. W. A. HAILES said that he was interested in this patient, as he had seen her in Melbourne. With regard to the value of periarterial sympathectomy, the results of this operation performed experimentally on the ears of albino rats had been published in *The British Journal of Surgery*. No alteration in the vascular condition of the part had been observed, but in spite of this experimental failure, the operation was worth a trial in clinical cases of the type of the patient presented.

DR. H. BOYD GRAHAM advanced the suggestion that the condition was one of scleroderma. In this condition it appeared that ganglionectomy promised more permanent results than periarterial sympathectomy. In support of this view he referred to a child presented at a recent meeting of the Melbourne Paediatric Society, suffering from bilateral scleroderma of the legs. Sympathetic ganglionectomy had been performed on one side, followed by very definite improvement in the condition on that side; and it was proposed to repeat the operation on the other side.

Branchial Cyst.

DR. ARMSTRONG's second patient was a man, aged eighteen years, who presented himself on August 5, 1930, complaining of a lump below the angle of the right jaw, which he had become aware of accidentally about six weeks before. It was painless, with only a sense of stiffness on extreme movements of the neck. On examination, in the upper part of the carotid triangle and partly beneath the anterior border of the sterno-mastoid muscle, was found a discrete, firm, roundish tumour with a vague sense of fluctuation,

¹ This patient died on October 20, 1930, following an attack of acute pulmonary oedema of six hours' duration.

apparently about two centimetres in diameter, with a slight degree of mobility in all directions. No glands could be detected here or elsewhere. No intrapharyngeal nor dental focus was detected. Previous history and family history were clear. The Wassermann test gave no reaction. A provisional diagnosis of branchial cyst was made and on September 30, under ether anaesthesia induced by the intratracheal method, operation was performed and the cyst presenting between the carotids was dissected deeply and removed.

DR. H. DOUGLAS STEPHENS said that one possible diagnosis was hydatid of the neck, which had proved to be the nature of a similar cyst which he (Dr. Stephens) had removed from a patient's neck recently. If not a hydatid, then he considered it must be a branchial cyst.

Perinephric Abscess with Nerve Signs.

Dr. Armstrong also showed a man, aged twenty-one years, who was admitted to hospital on August 30, 1930. Ten days prior to admission, whilst working in water up to his knees, he noticed aching pain in the left lumbar region, worse when he bent down. He came home and went to bed; pain did not ease with rest, but remained of a nagging, aching character, with no radiation. During the next few days he noticed he could not flex his spine in the lumbar region, though lateral or backward bending did not increase pain. On examination his temperature was 38.3° C. (101° F.) and his pulse rate 100 per minute. Tenderness was present in the left costo-vertebral angle and he was unable to flex his lumbar spine, though lateral and backward movement was normal and did not increase pain. All other examination failed to reveal abnormality. Six days from the onset, the patient woke up with numbness in both feet and inability to move the toes or feet. At the same time he complained of aching pains down the backs of the thighs and buttocks and the outside of the legs. The numbness and paralysis extended upwards symmetrically and he complained of numbness of both buttocks. Two days later there was sudden retention of urine and he noticed weakness of the anal sphincter. There was anaesthesia of both feet below the ankles and a saddle-shaped area on the buttocks. Knee and ankle jerks and plantar reflex were absent. He could flex the thighs and partially extend the legs. Foot-drop was present. X ray examination of the spine revealed no abnormality. For six days after admission his condition remained much the same, except for gradual recovery of power in the quadriceps extensor muscles and some improvement in sensation. A left perinephric abscess was then drained. Later a subcutaneous abscess to the left of the dorsal spine and continuous with the original abscess required incision. His temperature, which was 39.4° C. (103° F.) before and for three weeks after the operation, had become normal and the signs of nervous involvement were slowly disappearing.

DR. VICTOR HURLEY said that the case presented two problems, one concerned with the abscess and the other with the neurological signs. The signs of nervous lesion were certainly spinal in type and some confirmation of this was lent by the fact that the later skiagram showed a track running up to the upper part of the thoracic portion of the spine. The patient had had a boil on his neck some weeks before his illness and the condition had probably commenced as a bone infection at one of the vertebral epiphyseal lines in the thoracic region, with a secondary perinephric abscess. Dr. Hurley recommended that a further skiagram should be taken of the upper part of the spine and also a lateral view of the affected vertebra.

DR. H. F. MAUDSLEY said that the neurological signs present at the time of the meeting bore out Dr. Hurley's opinion and suggested pressure on the spinal cord rather than a true *cauda equina* lesion.

DR. W. A. HAILES expressed a doubt as to whether the original osteitis had really been high up in the thoracic region, and suggested that there had possibly been two foci, one in the thoracic part of the spine and one in the sacral region, causing the mixed signs of spinal cord and *cauda equina* pressure, with the latter clearing up and leaving the spinal cord signs predominant at the present time.

Aneurysm and Tumour of the Suprarenal Glands.

DR. A. L. BENNETT showed a man, aged fifty years, who had been ill for three weeks prior to admission to hospital on September 8, 1930. His illness commenced with headache, anorexia and weakness. Apart from a temperature of 37.8° C. (100° F.) at the onset, examination revealed no abnormality except for his cardiac condition. On the day of admission to hospital he complained of pain (not severe) in the lumbar region and had had slight nocturnal frequency of micturition for the previous three days. The pain and frequency then cleared up and did not recur. Examination of urine on this occasion revealed clumps of bacilli, but subsequent examinations all gave normal results, except for a few hyaline casts. There was slight loss of weight about this time. The sight in the left eye had been defective. About five years previously he had been examined for admission to a lodge and a cardiac condition in every way similar to his present condition was found. At that time and until the onset of the present illness, he had been working as an orchard labourer with no disability. Seventeen years previously he was in another hospital for several weeks with pyrexia for which no cause was found. The family history was clear, his wife and seven children being healthy.

On admission his temperature was 38.3° C. (101° F.), his pulse rate 96 and respiration rate 24 per minute. His blood pressure on the left side was 105 millimetres of mercury systolic and 58 millimetres diastolic; on the right side the figures were 100 and 60 millimetres respectively. (His systolic pressure while in hospital varied from 88 to 96 millimetres of mercury.) The cardiac apex beat was in the seventh left intercostal space 11.25 centimetres (four and a half inches) from the middle of the sternum. Extending about five centimetres (two inches) to the right of the sternum from below the right clavicle to the third intercostal space there was an area of dullness with a thrill and visible pulsation that was thought to be expansile. Very loud apical systolic and aortic systolic and diastolic murmurs were present. Murmurs were heard over the left side of the chest posteriorly. The liver dullness was increased 2.5 centimetres (one inch) below the costal margin; otherwise the abdomen was clear. Both pupils were equal in size and reacted to light and accommodation; the right pupil was slightly irregular in outline. The patient was blind in the left eye. X ray examination of the chest revealed a very large heart, with slight increase in size of the aorta in a lateral direction with lateral pulsation and pronounced right cardiac dullness. Attempts at blood culture, the Wassermann test, the Widal test and examination for *Bacillus abortus* all yielded no results on two occasions.

After admission to hospital the patient remained in much the same condition, with temperature ranging from 37.8° to 39.4° C. (100° to 103° F.) and a pulse rate of from 88 to 120 per minute. During the few days preceding the meeting he was very slow in responding to questions and appeared to be slightly deaf on the left side. Examination of the ear revealed no abnormality. There was no pain, cough, dyspnoea, oedema or alteration in voice; no obvious hæmorrhage had occurred and no abnormal condition of alimentary and urinary systems was present.

Dr. Mark Gardiner examined the patient's eyes and found that his blindness was due to hæmorrhage.¹

DR. S. O. COWEN said that the case was an interesting one. There were two aspects from which it had to be considered: first, as regards the heart condition, the fact that the man had worked for the last five years with fair efficiency was in favour of its being rheumatic rather than syphilitic. Although clinical signs suggested aneurysm, the X ray and other findings did not certainly confirm this. The pyrexia, anaemia, the generalized superficial gland enlargement with enlarged spleen and liver could best be explained by a diagnosis of lymphocytoma—a lymphosarcoma or Hodgkin's disease. The deafness might be explained by the infiltration of abnormal cells locally

¹This patient died on October 18, 1930. At post mortem examination an aortic aneurysm was found; bilateral tumours of the suprarenal bodies were present and two cysts were found arising from the anterior parts of the lateral ventricles of the brain.

or by hæmorrhage. Dr. Cowen said that the diagnosis might be clinched by the excision and microscopical examination of an affected gland.

DR. WILFRED FORSTER said that he considered Hodgkin's disease unlikely owing to the long history and a similar attack seventeen years before. He favoured a diagnosis of subacute bacterial endocarditis.

DR. IVAN MAXWELL said that in the main he agreed with Dr. Cowen, but that the basis was probably syphilitic. He considered that there were definite signs of an aneurysm, there being expansile pulsation in the second interspace. The vocal cords had not been examined, but it was possible that there was a mediastinal tumour as well as an aneurysm.

DR. MARK GARDNER reported that examination of the fundi revealed a pale retina on the right side, with dilated retinal veins, and on the left a considerable vitreous hæmorrhage, but no neoplasm.

MEDICO-POLITICAL.

DURING a recent official visit to the Western Australian Branch of the British Medical Association the Editor of THE MEDICAL JOURNAL OF AUSTRALIA invited the Council of the Branch to forward at regular intervals for publication in the journal a short account of its activities which might be of use to members of the Western Australian Branch and of interest to other Branches. The following statement has been received. Invitations of a similar nature have been sent from time to time to other Branches; it is hoped that they will follow the example of Western Australia.

Workers' Compensation Act.

In the present *Workers' Compensation Act* there are many difficulties and anomalies, and it is proposed by the Government to amend the Act at an early date. The Council has offered its assistance to the Government in this direction to endeavour to bring the working on to a better footing. With this end in view the Council, in conjunction with the Special General Committee at present reviewing all medical accounts, the reasonableness of which may be questioned by insurance companies under the Act, are formulating certain general principles which are considered essential to the successful working of the Act from a medical point of view. As a further contribution and expression of good will, a revision of the schedule of fees to be considered as a guide under the Act has been made and passed by a general meeting of members. In this certain anomalies have been removed and members have generously recognized the principle of reduction in fees where considered higher than would reasonably be charged privately a member of the industrial community, should he not be treated under the *Workers' Compensation Act*. The matter is still receiving consideration pending further action by the Government.

Model Lodge Agreement.

Owing to the financial depression, the Council of the Friendly Societies approached the Branch Council to consider the question of unemployed members of lodges.

The friendly societies proposed to keep on the medical lists all married members and single members with dependants who were unemployed and keep them financial by drawing on friendly societies' sick and funeral benefits and levying a further charge on members who were not out of work. They appealed to the medical profession to contribute 10% of their quarterly cheques to this unemployment fund. The Special Lodge Medical Committee and the Council, after careful consultation with the Council of the Friendly Societies, urgently recommended this assistance to the societies and the proposal was passed by general meeting, to come into force as from March 1 or April 1, 1931, whichever was the quarter night of the societies.

Local Medical Associations.

Whilst realizing that Western Australia is a large State in which medical men are often separated by many miles from one another, yet it was thought that "local medical

associations" might be formed on somewhat the same lines as at present followed out in the Eastern States. The success of such ventures rests really in the enthusiasm displayed by country members, and already we are receiving most encouraging replies from members in areas which appear to be suitable for their formation.

Honorary Medical Service to Public Hospitals.

The Branch views with grave concern the growing abuses of the system of honorary medical service to public hospitals at present in operation, and has appointed a subcommittee to review the whole position and report back to the Branch the results of their investigations in two months' time.

Model Rules Governing Procedure in Ethical Matters of a Branch in Australia.

The revised model rules governing procedure in ethical matters were approved of by the Branch and are to be incorporated in the rules.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Grant, Alan Mostyn Bradford, M.B., B.S., 1929 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned has been registered under the provisions of the *Medical Act*, 1928, of Victoria, as a duly qualified medical practitioner:

Thorne, Henry Joseph Robert, M.B., B.S., 1923 (Univ. Melbourne), c.o. Agent-General for Western Australia, Savoy House, Strand, London.

Obituary.

JESSIE JARDINE LANG.

WE regret to announce the death of Dr. Jessie Jardine Lang, which occurred at Mosman, New South Wales, on June 16, 1931.

JOHN LAING MARTIN MCCREADIE.

WE regret to announce the death of Dr. John Laing Martin McCreadie, which occurred on June 22, 1931, at Sydney, New South Wales.

ALFRED ELAND SHAW.

WE regret to announce the death of Dr. Alfred Eland Shaw, which occurred at Sydney, New South Wales, on June 16, 1931.

Correspondence.

PROFESSIONAL ADVERTISEMENT.

SIR: The letters of Dr. Keith Barry and Dr. H. Burnett Bruce, which appeared in the journal of May 16 and June 6 respectively, commenting on the action of the

British Medical Association (New South Wales Branch) in passing certain resolutions *re* professional advertisements, call for an answer.

One feels that a good deal of criticism that is levelled at the British Medical Association is due to lack of knowledge.

Within the last few years it has been my good fortune to occupy various official positions in the local association to which I belong, and I have thus gained an inside knowledge of the enormous amount of work done by the Council. I wish it to be understood, however, that the views I put forward are purely my own and not those of any official body.

With regard to the contention of Dr. Barry, for the use of the ballot box, in which he is supported by Dr. Bruce, I can only say that neither of these gentlemen has had much experience of this method where medical men are concerned. Time and time again the response has proved the method to be hopeless. It is only by discussion of resolutions that the arguments for and against can be fully ventilated. The fact that less than fifty members were present at the general meeting, and most of them not general practitioners, is not the fault of the Association or its system, but that of the members themselves, who show an utter disregard for the welfare of their fellow practitioners and their own selves. It appears that the only capability of a number of these absentees is to offer criticism, and criticism which is rarely constructive.

Dr. Bruce asks why he cannot inform his old patients that he has resumed practice. There is nothing in the by-laws which says he must not do so. The only restriction is that he shall not insert any advertisement relating to his profession or the practice thereof. Dr. Bruce can certainly communicate personally with those patients who were under his immediate care prior to his absence from practice, but note that I have said his immediate care, which does not mean all his former patients. No medical practitioner has any prescriptive right over any patient—his patient of yesterday is not his patient today. Unfortunately for himself, Dr. Bruce goes too far. He wishes to inform his prospective patients. To my way of thinking, and I can see no other interpretation, this is advertising for patients—the strongest argument in favour of the restriction of professional advertisements in the public press.

Would it not be advisable before quoting examples of medical men who have their portrait published in the press, as the result of their outside interests, such as politics *et cetera*, to consult the list of members of the British Medical Association? I think it would repay both these gentlemen. It must be remembered that the Association is but a private body, which can discipline its own members only and not those practitioners outside of it.

Both Dr. Barry and Dr. Bruce are of the opinion that there are many other matters with which the Association might concern itself. I fear that if either of these gentlemen knew the enormous amount of work being done by Council, they would be agreeably surprised. The work resulting from the *Workers' Compensation Act* alone is enormous.

Now for this last mentioned subject. Dr. Bruce is quite correct in his statement that it is only at the request of the employer or employee that the injured worker can be examined by a referee. If, however, the employer, and this is the general rule, delegates his rights to the insurer, has the Association any power or right to interfere? The regulations to the Act state clearly that the employer shall not, without the written authority of the insurer, incur any expense of litigation or make any payment, settlement, or admission of liability in respect of an injury to, or claim made by any worker. The result is the employer is for all practical purposes the insurer. Hence the reason why the insurance companies require the injured worker to be examined by their medical practitioner.

Time there was when I opposed Schedule D very strongly, but experience has proved to me—and I think I am supported in this by the majority of the profession—that we have not fared badly. Dr. Bruce is still entitled to receive his full fee from his patient, but will his purse be as full? I think not, especially in these times of financial upheaval.

I feel sure that not for one moment would he contemplate a return to the period prior to the introduction of

Schedule D. Moreover, I am told on good authority that had Council not come to some arrangement with the insurer, a schedule would have been laid down which would not have been nearly so generous or satisfactory to the profession as Schedule D. Of course, it is open to Dr. Bruce to produce some other and better method of dealing with cases arising under the *Workers' Compensation Act*, which I am sure, Mr. Editor, you will be only too pleased to publish.

Dr. Barry has raised a subject of paramount importance, namely, the affiliated local associations. Might I suggest, not only to these two gentlemen, but to all members of the Branch, general practitioners and otherwise, that they join their local associations, if they are not now members, and not only join, but take an active part therein. These local associations meet regularly, discuss various matters of medico-political interest *et cetera*, and forward motions to the annual meeting of delegates to the local associations with Council. They offer what the British Medical Association will always welcome—constructive criticism—and not the arm-chair criticism of work done by the Council, by members unaware of the facts or the work being done.

Let it be remembered that the activities of an association are not only those of its governing body, but the activities of each and every individual member.

Finally, to Dr. Bruce I would point out that the *pièce de résistance* of Dr. Barry's letter, the *post scriptum*, is most clearly answered by Dr. C. H. E. Lawes in his letter published in the journal of June 6.

I thank you, Mr. Editor, for allowing me to take up so much of your space.

Yours, etc.,

HUGH HUNTER.

"Westgate,"
17, Oxford Street,
Waverley.
June 11, 1931.

"AVERTIN" IN SURGERY AND OBSTETRICS.

SIR: We wish to comment on the quotation made on our experimental study of "Avertin" and "Sodium Amytal" by Dr. Cecil Coghlan in his article, "Avertin in Surgery and Obstetrics," which appeared in the issue of this journal on June 20, 1931, page 737.

Unfortunately this author has overlooked the obvious fact that the mistake is wholly a printer's error, and has already been editorially corrected in a subsequent issue (May 3, 1930, page 598), so that the quotation should read:

"'Avertin' is a reasonably safe and non-toxic anaesthetic, but as with human beings the margin of absolute safety is somewhat less than has been claimed by some of the earlier observers" *et cetera*.

We compared our experimental results, therefore, with the experience of many clinicians who have concluded that the higher doses, for example, over 0.15 gramme per kilogram administered by Kirschner and others, are unsafe for routine administration.

Yours, etc.,

ADOLPH BOLLIGER.
KEMPSON MADDOX.

Royal Prince Alfred Hospital,
Camperdown,
June 24, 1931.

BRANCH SUBSCRIPTIONS.

SIR: At the Brisbane Congress in 1899 I suggested the foundation of an Australian Medical Association. My primary object in the long and tedious fight to establish THE MEDICAL JOURNAL OF AUSTRALIA was the hope that it would be a powerful instrument to bring about such an association.

The journal cost to English members is one-tenth of their subscriptions. In Victoria ours is five-tenths. I

believe I am one of the few who regularly read *The British Medical Journal*. I regard it as sinful that nearly £5,000 per annum plus rate of exchange is sent out of Australia, mostly for purposes of an ill-placed sentiment that has nothing to do with the binding or loosening of the Imperial tie.

Our own journal would largely increase its advertisements if *The British Medical Journal* were to cease circulating to every member in Australia.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,
Melbourne,
June 13, 1931.

AN EXPLANATION.

SIR: In reference to pamphlet on "Neo Rhomnol," in which the name of Dr. Lindsay Dey, of Sydney, is mentioned, we wish to state that no authority was received from Dr. Dey for the use of his name, and we have, at the doctor's request, discontinued the issue of this pamphlet.

Yours, etc.,

STUART ALEXANDER AND COMPANY.

7, Spring Street,
Sydney,
June 18, 1931.

Books Received.

MIDWIFERY FOR NURSES, by Douglas Miller, M.D., F.R.C.S., M.R.C.P.; 1931. London: Edward Arnold. Crown 8vo., pp. 263, with illustrations. Price: 6s. net.

THE HISTORY OF MEDICINE, A SHORT SYNOPSIS, by Bernard Dawson, M.D., F.R.C.S.; 1931. London: H. K. Lewis. Crown 8vo., pp. 174, with illustrations. Price: 7s. 6d. net.

THE PHYSICAL AND RADIOLOGICAL EXAMINATION OF THE LUNGS, WITH SPECIAL REFERENCE TO TUBERCULOSIS AND SILICOSIS, INCLUDING A CHAPTER ON LARYNGEAL TUBERCULOSIS, by James Crockett, M.D., D.P.H., F.R.C.P.E.; Second Edition; 1931. London: H. K. Lewis. Demy 8vo., pp. 306, with 151 illustrations, including 40 plates. Price: 16s. net.

Diary for the Month.

- JULY 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JULY 10.—Queensland Branch, B.M.A.: Council.
JULY 14.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JULY 22.—Victorian Branch, B.M.A.: Council.
JULY 24.—Queensland Branch, B.M.A.: Council.
JULY 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 30.—South Australian Branch, B.M.A.: Branch.
JULY 30.—New South Wales Branch, B.M.A.: Branch.
AUG. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 5.—Victorian Branch, B.M.A.: Branch.

Medical Appointments.

Dr. C. de Monchaux (B.M.A.) has been appointed Radio-therapist to the Dunedin Hospital, New Zealand.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xvi.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Medical Superintendent.

RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Honorary Physician, Honorary Anaesthetist.

ROYAL AIR FORCE, MEDICAL BRANCH: Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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